MAY 10 1927

VOLUME 17 NUMBER 4

APRIL, 1927

Phytopathology

An International Journal

Official Organ of the

American Phytopathological Society

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Price { $8.00 per year, United States and dependencies, Mexico and Cuba
$8.25 per year, Canada
$8.50 per year, elsewhere

PUBLISHED MONTHLY BY THE SOCIETY
OFFICE OF PUBLICATION
Cor. Lime and Green Streets, Lancaster, Pa.

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HAROLD WAKEFIELD FITCH

PHYTOPATHOLOGY

VOLUME 17 NUMBER 4

APRIL, 1927

HAROLD WAKEFIELD FITCH

1897–1926

L. M. MASSEY

Harold Wakefield Fitch was born at Claremont, New Hampshire, June 5, 1897, and died at Grand Rapids, Michigan, May 16, 1926. After his graduation from the Claremont High School in 1916, he entered New Hampshire University, from which he received the degree of Bachelor of Science in 1921.

During the summer of 1920 Fitch served as Special Field Assistant in Albany County, New York, in which position he demonstrated marked ability in handling the applied phases of fruit disease control. In April, 1921, he was appointed to the Herman Frasch Fellowship in Plant Pathology at Cornell University and held this appointment until February 28, 1925, when he resigned to accept a position on the scientific staff of the Niagara Sprayer Company, with the privilege of returning to Cornell during the winters to work for his doctor's degree, for which he had practically completed the requirements at the time of his death. In this last position he had charge of demonstrational dusting work in several mid-western states, with headquarters at Grand Rapids, Michigan.

At Cornell, Fitch was engaged in testing the relative merits of dusts and sprays in the control of fruit diseases and he contributed much to our knowledge of this subject. His interest lay primarily in the applied field. He had a personality that inspired friendship and confidence, and a tireless energy that enabled him to conduct the extensive tests necessary in the arduous task of the orchard-testing of sprays and dusts. His characteristic devotion to his work, even to the neglect of his own health, was no doubt an important factor contributing to his untimely death.

His two papers, "Some results of dusting experiments for apple scab and peach leaf curl in 1921–22," published in Proc. N. Y. State Hort. Soc. **68**: 42–60. 1923, and "Quantitative determinations of sulfur fungicides on foliage," published in Phytopathology **15**: 351–354. 1925 (revised, Phyto-

pathology **16**: 427–428. 1926), are his most important contributions to the literature of plant pathology.

Never so rushed with work that courtesy was forgotten, never too busy to take the time that was necessary to offer sincere and sympathetic help to the grower with his practical problem, always pleasant and optimistic, with an enthusiasm for whatever he might undertake that completely overcame the monotony of routine tasks, Fitch endeared himself to all with whom he was associated.

A NEW SPECIES OF EXOBASIDIUM

J. W. Hotson

INTRODUCTION

The examination of the genus *Exobasidium* reveals a great need for further cultural study of the species referred to it. The fact that the members of this genus are obligate parasites, or at least grow poorly on nutrient media, makes it very difficult to decide without host inoculation whether the various species listed are autonomous species or merely biologic forms. A careful and detailed account of the morphology and life history of *Exobasidium vaccinii* has been given by Woronin (10). This description has been based on observations extending over at least two seasons. Since his time, however, little attention has been paid to the morphology, although various new species have been described largely on the basis of form and color of the hypertrophy on new hosts or on the basis of a single or limited observation of an individual gall. It is quite probable, therefore, that there are some forms named which are not autonomous species. Of late years an effort has been made to rearrange these forms, grouping them mainly on morphological characters. Looking toward this end, E. Rostrup, in his Danish Fungi (4, pp. 350–352), divides the different types of *Exobasidium* in Europe into three morphologically distinct groups.

(1) The circumscribed type, which has its fruiting area on limited spots on the leaves, forming irregular gall-like bodies, each basidium giving rise to four small spores, 5–8 × 1–2 μ. Apparently *E. vaccinii* (Fuck.) Wor. has been taken as the type for this group.

(2) The penetrating type, in which the mycelium penetrates either the whole host plant or single branches, causing hypertrophies on them and producing witches'-brooms, each basidium bearing only two large spores, 25–32 × 8–12 μ.

(3) The Arctostaphylos type, intermediate between the other two, which occurs only on *Arctostaphylos*. This type is illustrated by *E. arctostaphyli* Harkn. with spores 12–17 × 3–5 μ.

In the first group, Rostrup has apparently taken the spore measurements of *E. vaccinii* as given either in Saccardo's Sylloge Fungorum (5) or in Winter's Die Pilze (8), both of which, as has already been pointed out by Richards (3), are incorrectly reported. Instead of 5–8 × 1–2 μ, the measurements should read 14–16.8 × 2.8 μ (10), or, as it is sometimes put, 14–

17 × 3 μ. On the basis of spore measurements, this correction would eliminate Rostrup's Arctostaphylos type and combine it with *E. vaccinii*.

Again in the second division the measurements of the spores should be, as explained by Burt (1, p. 655), 16–20 × 7–8 μ, which corresponds to the size of the spores of the American collections of *E. vaccinii uliginosi*.

In America, Burt (1) has reduced the eleven species described as occurring on this continent to three autonomous species, the others being synonyms of one or another of these three. He sees no evidence that any of them are biologic forms. These three species are separated on purely morphological lines; the type of the hypertrophy and the host plays little or no part in the determinations. The first species, *E. vaccinii* (Fuck.) Wor., embraces all those forms in which the measurements of the basidiospores come within the limits recorded for this species, 14–17 × 3 μ. This is the common and widespread species found in practically all parts of the United States where ericaceous plants grow. As synonyms of this species Burt has placed the following: *E. azaleae* Peck (*E. discoideum* Ell.), *E. rhododendri* Cramer, *E. peckii* Halst., *E. andromedae* Peck, *E. cassandrae* Peck, *E. arctostaphyli* Harkn., *E. cassiopes* Peck, *E. oxycocci* Rostrup, *E. karstenii* Sacc. and Trott (*E. andromedae* Karst, non Peck), *E. vaccinii myrtilli* (Fuck.) Juel. Of these the following have been reported from the state of Washington: *E. vaccinii* on *Vaccinium membranaceum*, *V. nevadensis* and *Rhododendron albiflorum*; *E. arctostaphyli* on *Arctostaphylos uva-ursi*; *E. cassiopes* on *Cassiope mertensiana*; *E. oxycocci* on *V. intermedium*; *E. vaccinii myrtilli* on *V. deliciosum*.

The second species, *E. vaccinii uliginosi* Boud., corresponds to Rostrup's second group and is distinguished by the fact that each basidium has only two spores, 16–20 × 7–8 μ. This species has been collected on Mount Rainier by C. V. Piper (Acc. No. 443).

The third species is *E. symploci* E. and M., in which conidia only are known. It occurs on *Symplocus tinctoriae*, and has not been reported from Washington.

Burt has also found such a close resemblance of the morphological characters to those of *Corticum* and *Peniophora* that he has followed Saccardo's example and placed this genus in the Thelephoraceae rather than raising it to the rank of an order as Hennings has done in Engler and Prantl's "Die Natürlichen Pflanzenfamilien."

DISTRIBUTION AND HOSTS

The *Exobasidium* under consideration was first found in the spring of 1917 in the vicinity of Seattle, Washington, on *Vaccinium parvifolium* Smith. Since then it has been collected on the same host on Bainbridge

Island, Washington, by the writer; on Whidby Island, Wash., by Lena Hartge; near Snoqualmie Falls, King County, Wash., by Howard and Charles Gray; in the Elwha Valley, Olympic Mountains, Wash., by E. Hopf; and on Alsea Mountain, Oregon, by S. M. Zeller, and again by C. E. Owens. On April 25, 1926, it was collected by T. C. Frye near Snoqualmie Pass, Wash., on *V. ovalifolium* Smith.

The observations on this fungus up to 1921 were reported before the Pacific Division, American Phytopathological Society (2).

THE GALL AND APPENDAGES

During the first year of infection the stems are only slightly hypertrophied, but gradually increase in size from year to year, eventually forming a well pronounced gall (Fig. 2). The galls are firm and made up of hypertrophied host tissue with the perennial mycelium growing between the

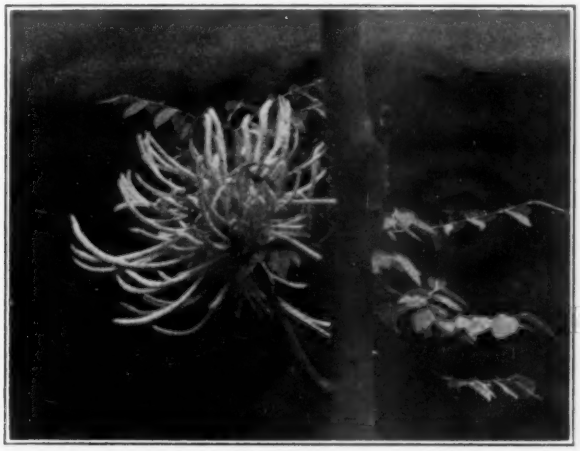

FIG. 1.—Mature appendages of a gall on *Vaccinium parvifolium* infected with *Exobasidium parvifolii*.

cells. They seldom completely girdle the stem so that the branch is not usually killed immediately. The longevity of the parasite is intimately related to that of the host branch on which it grows. The death of the host branch is immediately followed by the death of the fungus, there being no evidence that the mycelium lives as a saprophyte on the dead branch.

Each spring, in Seattle, about the middle of April, or a little earlier in some seasons, large numbers of soft, fleshy, more or less horn-shaped processes or appendages grow out from all parts of this gall (Fig. 1). These are usually single, occasionally branched, elongate, clavate, or more or less cylindrical in form (Plate IX, A). As they grow they pass through various

shades of pink, sometimes reaching a spectrum red (Ridgway). Later they become light green (Scheele's Green, Ridgway) without any evidence of the fungus on the outside. These appendages vary in length from 2 to 10 cm. and occasionally are as long as 15 cm.; the diameter is from 3 to 4 mm. or sometimes more toward the tip. The number of appendages depends upon the age of the gall. Over a hundred of these outgrowths have been counted on a single gall, while in another instance a single appendage was produced as the result of an infection at a leaf-scar. As these green processes approach maturity, which is early in June, they pass through various shades of color somewhat similar to those exhibited in their earlier stages of development. These changes begin at the tips and gradually work back to the base, the appendages eventually becoming completely white and often powdery (Fig. 1). Early in June, seldom later than the middle of the month, the appendages begin to turn dark and dry up, and in a couple of weeks all the beauty has disappeared and nothing but a few dark, thread-like strands are attached to the gall (Fig. 2, B). Thus from about the middle of June throughout the rest of the season the galls are so inconspicuous that they are easily overlooked.

STRUCTURE OF THE APPENDAGES

A microscopic examination of these fleshy processes shows that they are composed of the tissue of the host with the mycelium of the fungus growing between the cells. They are clearly outgrowths of the host, which has been stimulated to produce new cells by the action of the parasite. A comparison of one of these processes with a normal shoot shows a number of striking differences (Plate IX, B and C). In the first place, the shape is different. The normal young twigs of *V. parvifolium* are more or less four-sided with short spines over the surface, while the appendages are much smaller in diameter, more cylindrical in form, and without spines. There is also a marked difference in the color of the two. If cross sections are compared, it will be seen that the normal stem has one continuous ring of cambium, while in the appendage the vascular tissue is broken up into a number of more or less distinct parts (Plate IX, B and C). Moreover, in the appendages a partial ring of secondary bundles is produced in the cortex just below the epidermis, which, it may be noted, is not as regular or as definite as in the normal twig. By comparing B and C of the plate these points may be seen, as well as the differences in the general character of the cells that make up the ground tissue of each. It should be borne in mind, however, that the cross sections do not give the relative size. In reality the diameter of an appendage is much smaller than that of the normal shoot.

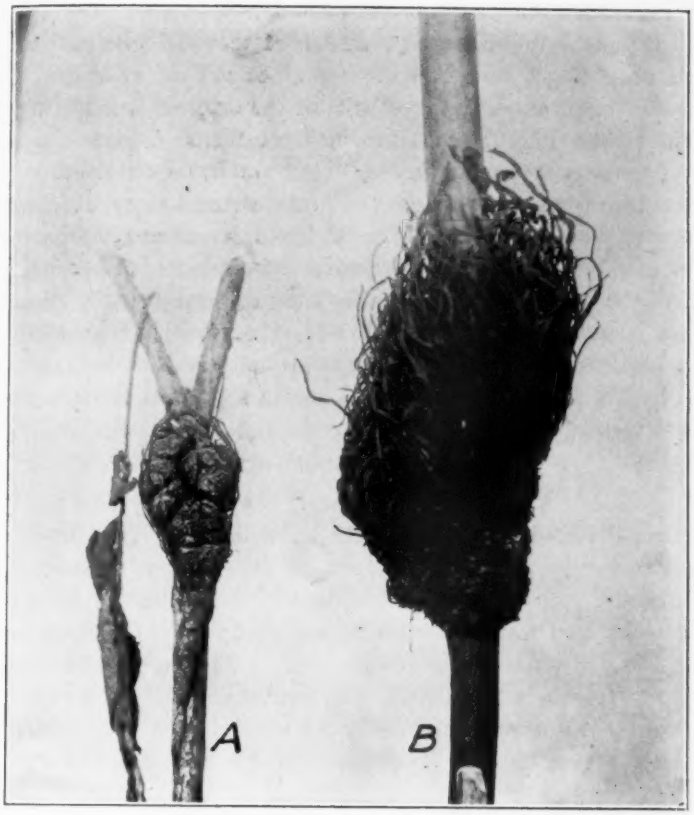

FIG. 2.—A. A gall with the appendages removed. B. A gall with only a few of the appendages removed.

THE MYCELIUM

The mycelium is perennial and intercellular in the cortex of the host. As the appendages grow out in response to the stimulation of the fungus, the mycelium keeps pace with them. The hyphae are very fine, about 1.5 μ in diameter, and much branched. When these processes approach their maximum growth, the hyphae grow between the epidermal cells, here and there sending out branches that swell up, forming basidia which eventually produce basidiospores. The mycelium may live several years in the host. One gall has been observed to produce appendages for seven years. It would appear that at times the mycelium in the gall remains somewhat dormant, not producing sufficient stimulus to induce appendages to grow. In one instance, in the early spring of 1919, a certain gall failed to produce appendages and was considered dead. The next spring (1920), however, ap-

pendages began to grow out as usual, although in smaller numbers. In the spring of 1921 still fewer were produced, after which the gall apparently died as no appendages have been formed since. This, of course, may have been a case of reinfection, the mycelium of the original gall having died out during the winter of 1919 and the host reinfected during the spring or summer of the same year, but in that case the normal condition would have been to produce fewer appendages the first year and more the second—just the reverse of what happened. The evidence favors dormant mycelium as the better explanation. Moreover, as the infected branch becomes less and less vigorous the vitality of the parasite is correspondingly reduced. In this dying process it is quite possible that a point is reached where the mycelium does not produce sufficient stimulus to cause the appendages to grow but by the following year would regain a portion of its vigor.

BASIDIA AND BASIDIOSPORES

The basidia are formed by lateral branches of the hyphae pushing their way between the epidermal cells of the appendages and swelling. At first these project only slightly beyond the host cells, but at maturity often extend half their length. This portion, being relieved of the pressure of the epidermal cells, sometimes enlarges rather abruptly. The diameter ranges from 7 to 10 μ. The basidiospores are usually four on each basidium, sometimes five. They are elongated, non-septate, usually somewhat curved, finely granular, measuring 11–20 × 2.5–4 μ (Plate IX, E). Although the basidiospores are unicellular when mature, the first step toward germination is the formation of septa, usually three in number but not constantly so (Plate IX, F). No septate spores have been found attached to the basidium, but if left in water over night many of the spores become septate.

CONIDIA

Intermingled with the basidia but usually in large numbers are small acicular, one-celled conidia (Plate IX, G). These are formed singly at the ends of lateral branches but do not always inhibit the growth of the branch, so that occasionally they are found apparently lateral near the tip. The conidiophores often arise in tufts so that large numbers of conidia are formed in the same vicinity, giving a sort of mealy appearance to the horn-shaped processes. Whether or not these spores form part of the life cycle of the fungus under consideration could not absolutely be determined. They are, however, constantly, year after year, found associated with it on the same kind of mycelium, and under such intimate conditions that the writer has little hesitation in considering this spore-form as belonging to the life cycle of the *Exobasidium*. Inoculation experiments in which these conidia

were used failed to reproduce the disease. This, however, is not surprising, as the majority of experiments in which basidiospores and mycelium were used also failed. The mycelium producing the conidia can be traced back into the tissue of the host.

CULTURAL STUDY

Several attempts were made to grow the fungus on artificial media but with little or no practical results. Media made of potato, sugar, and stems of *V. parvifolium* were used, but no better results were obtained than Richards (3) got in his experiments with *E. andromedae*.

Better results, however, were obtained by making the cultures on living plants. In order that more galls might be obtained for future study inoculation experiments were made, first with *V. parvifolium*. Plants in a small ravine on Bainbridge Island were selected for this purpose. Deep in this ravine it is quite moist even in July and August. It was found that a very careful selection of host plants was necessary in order to insure success, as only those deep in the ravine and thus in a fairly damp situation became infected.

In the first experiments a few appendages containing mature spores were washed in sterile water, which was then put into an atomizer and sprayed over the whole plant. This method was not successful and was soon abandoned. The following method proved more successful. Portions of the appendages containing mature spores were applied to wounds in the host and then bound up, some with absorbent cotton, some without. These inoculations were made about the middle of June on (a) the current year's growth, (b) the last year's growth, (c) old stems, (d) leaves, and (e) roots. Of these, negative results were obtained on leaves and old stems; but infections were obtained on all the others, producing galls from which the characteristic horn-shaped processes grew out the next spring, few appearing the first spring, but the number increasing in successive seasons until the twig died or had lost so much of its vigor that the parasite died first, then the twig.

Having established the parasite on *V. parvifolium*, attempts were made to grow it on other species in the hope that it might produce some other kind of gall that might show its relationship to *E. vaccinii*. The available species for this purpose were *V. ovalifolium*, *V. ovatum* and *V. uliginosum*. Unfortunately *V. vitis-idaea* was not available for these experiments. Negative results were obtained in all attempts to inoculate *V. ovatum* and *V. uliginosum*. One typical gall was obtained on *V. ovalifolium*. As has been said, the fungus was later collected on this host near the Snoqualmie Pass, Washington.

DISCUSSION

As has been suggested, some species of *Exobasidium* have undoubtedly been described on too meager data, *i.e.*, on the characteristics of a single detached gall, or on observations for a single season without taking into consideration other forms of hypertrophy the parasite might produce. Obviously distinctive morphological characters are the most important factors in determining species, but they do not constitute the only characteristic to be considered. The relation of the species of *Exobasidium* to its host is comparable in some degree to that of a rust to its host. That is, the species of *Exobasidium* approach the condition of obligate parasites and not only the morphological characters but also the host and the effect on the host must be taken into account.

The size of the basidiospores of the *Exobasidium* on *V. parvifolium* come approximately within the limits given for *E. vaccinii*, but their shape is usually more cylindrical and rounded at both ends rather than spindle-shaped as reported for the latter species.

In searching the literature on this subject no description or illustration of a gall resembling this one has been found. The nearest approach to it is the "shoot gall" referred to by Burt (1, p. 629) which occurs on certain hosts of *E. vaccinii*. In this case, however, it is the lateral bud that grows out as a result of the stimulus of the parasite and produces a single gall, while in the species under consideration the gall does not necessarily arise from a bud but from any place where infection takes place, usually at some wound. Under the stimulation of the parasite the cambium is broken up and branches or shoots grow out, not one or two, but large numbers. The localization of the gall and the peculiar effect of the stimulus on the meristematic region of the host is unique. Moreover it is found on distinctly new hosts, and as far as is known it produces only one type of gall, there being no indication of hypertrophies of the leaves, buds, or flowers. For these reasons the writer is inclined to consider this fungus an autonomous species, and proposes the name *Exobasidium parvifolii* with the following description.

Exobasidium parvifolii n. sp.

Mycelium perennial, much branched, intercellular, about 1.5 μ in diameter, producing cankers only on the stem or young branches from which grow out numerous fungous-permeated, corniform processes, 2–15 cm. long and 2–4 mm. in diameter; basidia hyaline, mostly claviform, 7–10 μ in diameter, formed on the surface of the corniform processes; sterigmata 4, sometimes 5; basidiospores cylindrical, hyaline, non-septate but becoming septate on germination, often curved, 11–20 × 2.5–4 μ; conidiophores unbranched,

similar to the hyphae, often in tufts; conidia simple, usually terminal, small, acicular, 5.5 ×1.5 μ.

Hab. On the young stems (1–3 years old) of *Vaccinium parvifolium* Smith, in Washington and Oregon; on *V. ovalifolium* Smith, Snoqualmie Falls, Washington.

Mycelio perenni, ramoso, intercellularis, 1.5 μ in diam. circa; carcinomatibus tantum in ramis junioribus ex quibus multi corniformes ramuli exerescent intercurrentes inter cellos, 2–15 cm. longis et 2–4 mm. in diam.; basidiis hyalinis, plerumque claviformibus, 7–10 μ in diam., formatis in superficiss eorundem ramulorum; sterigmatibus 4, interdum 5; basidiosporis cylindraceis, hyalinis, saepius curvatis, 11–20 × 2.5–4 μ; conidiosporis simplicibus, verisimilibus hyphis, saepe eristatis; conidiis simplicibus plerumque terminalibus, parvulis, accularis, 5.5 × 1.5 μ.

Hab. In ramis junioribus *Vaccinii parvifolii* Smith, Washington et Oregon. U. S. Amer. bor. et *V. ovalifolii,* Washington, U. S. Amer. bor.

SUMMARY

1. A newly described *Exobasidium* occurs on the main stems or young branches of *Vaccinium parvifolium,* less frequently on *V. ovalifolium,* probably gaining access by some wound.

2. The mycelium is perennial, one gall having been observed for seven seasons, and several others from three to five.

3. According to careful observation for eight seasons, only the main stem, branches and roots are infected, with no indication of the disease on the leaves, buds, or flowers.

4. Large numbers of small, acicular, one-celled conidia are associated with this fungus.

5. The results of inoculation experiments thus far show that *V. parvifolium* and *V. ovalifolium* can be infected artificially and *V. ovatum* and *V. uliginosum* can not; that young branches up to three years old and roots are the only host parts infected; that the only kind of gall formed is that producing long horn-shaped processes; that these occur only in the spring from April to June inclusive; and that infection occurs most readily at wounds, even a slight wound such as a leaf scar being sufficient.

6. The name *Exobasidium parvifolii* sp. nov. is proposed and a formal description given.

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EXPLANATION OF PLATE IX

A. Horn-shaped appendages showing the infrequent branching.

B. Cross section of one of the appendages showing the arrangement of the fibro-vascular bundles.

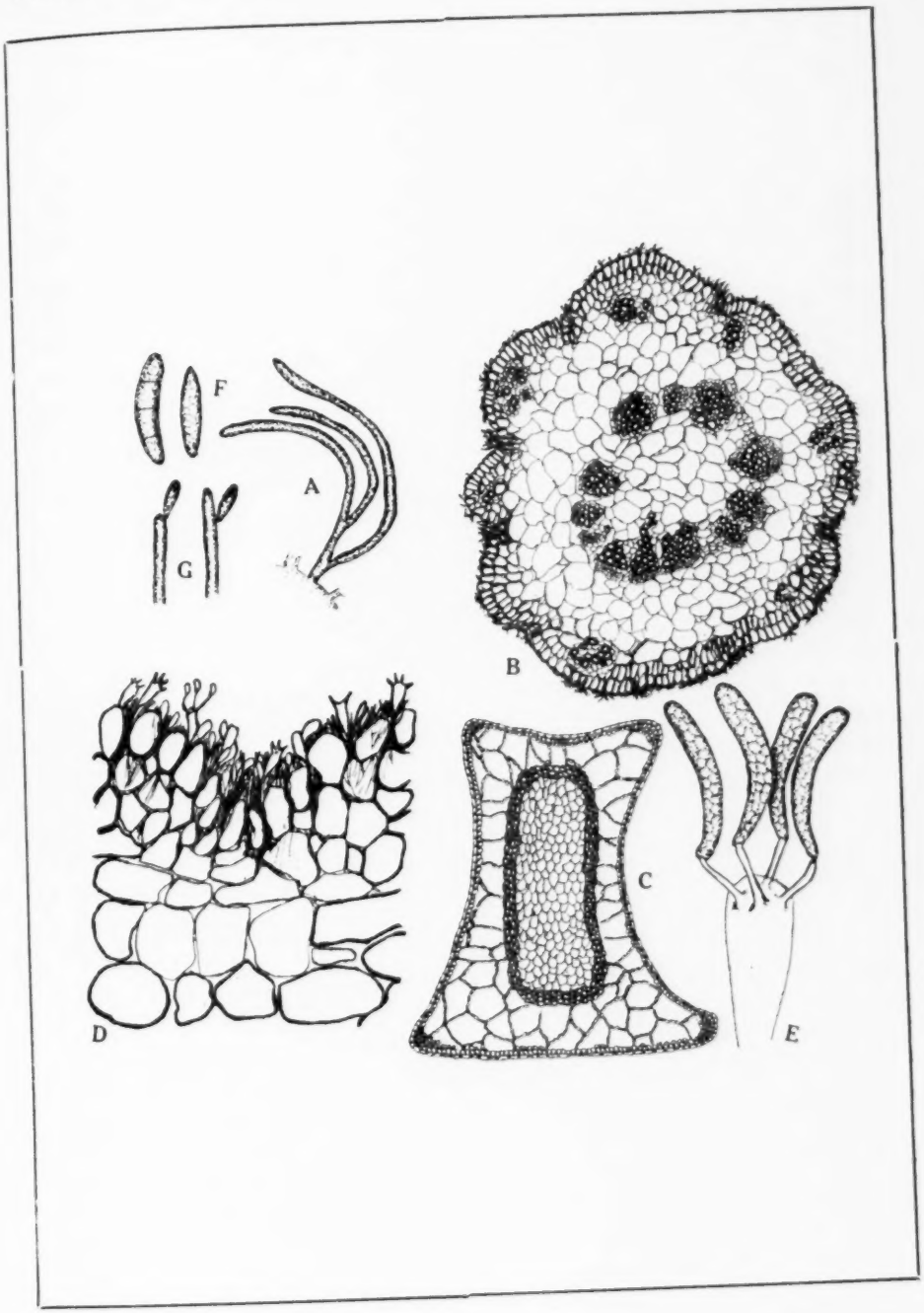
C. Cross section of a normal branch of *Vaccinium* much reduced.

D. A portion of one of the appendages showing mycelium, basidia and basidiospores.

E. A basidium with basidiospores.

F. Basidiospores.

G. Conidiophores with conidia.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PALE DWARF DISEASE OF PEANUT (*ARACHIS HYPOGAEA*)

CARL HARTLEY

During the course of an investigation of bacterial wilt of peanut for the Instituut voor Plantenziekten, Buitenzorg, Java, in 1921–22, a type of dwarfing was encountered of which no description has been found in the literature. The name "pale dwarf" has been applied to it in order to distinguish it from club-leaf dwarfing, in which the chlorophyll content is above, rather than below, normal, and which will be described in another paper.

SYMPTOMS

In the pale-dwarf plants the early development of roots, hypocotyl, and stipules is normal or nearly so. The first leaflets are pale, much shortened, and their width more reduced than their length. The different segments of the same leaf are not always equally affected. In general, opposite leaflets are very much alike, but the distal pair is sometimes more reduced than the basal. In the most seriously affected plants the petioles are also greatly reduced. Subsequent development of roots and stipules is subnormal, but only to the extent that would be expected as a result of the small carbohydrate production of the dwarfed top. Two extreme cases of the disease are shown in Plate X, A. The same abnormality, in a somewhat more typical form, is shown in Plate X, B. The habit of the plant is not greatly modified, the branches being no more ascending than are those of normal plants. The proliferation of leaves and stems reported recently in pale stunted plants in South Africa (1) has not been observed in the pale-dwarf plants.

In such cases as are shown in Plate X, A, the plant may live for a considerable time and put out successive pairs of tiny leaves, but is likely to die without recovery. Such a plant, at a later stage, is shown in Plate X, C, the photograph being taken 6 weeks after seed-sowing and just before the plant died. In cases not so severe, as shown in Plate X, D, and in the right-hand plant in Plate X, B, the leaves developed later are progressively more normal, until the plant is producing leaves entirely normal in size, shape, and color. Plants in which the leaflets of the first one or two pairs of leaves are one-half normal length and one-third normal width are soon scarcely distinguishable from normal plants. Such a plant as that in Plate X, D matures only a little later than the normal plants, but is small as a

result of the time lost at the beginning of the season, and produces few properly filled seeds. Between decidedly dwarfed plants and normal plants all gradations are found.

No curling or crinkling of the leaflets has been observed. With a single doubtful exception, the disease has never been found attacking a plant whose first leaves were normal. The typical course of the disease involves steady improvement. These characters distinguish the trouble very sharply from the African Kräuselkrankheit of Zimmerman (8, 9), which he says involves crinkling of the leaves, and from which a plant never recovers. While the Java krulziekte was described by Rutgers (4) as without crinkling of the leaves, it appears that cases of krulziekte often develop in plants which were normal in the juvenile stage; this, and the apparently deferred maturity and long continued flowering of the krulziek plants, argue against any relationship between Rutger's disease and the pale dwarf disease.

Dwarfing sometimes occurs as a result of infection with *Bacterium solanacearum*, but there is not the slightest possibility of confusing the bacterial dwarfing with the pale dwarf condition. The latter occurs both on wilt-sick fields and in plantings where the wilt bacteria do no damage.

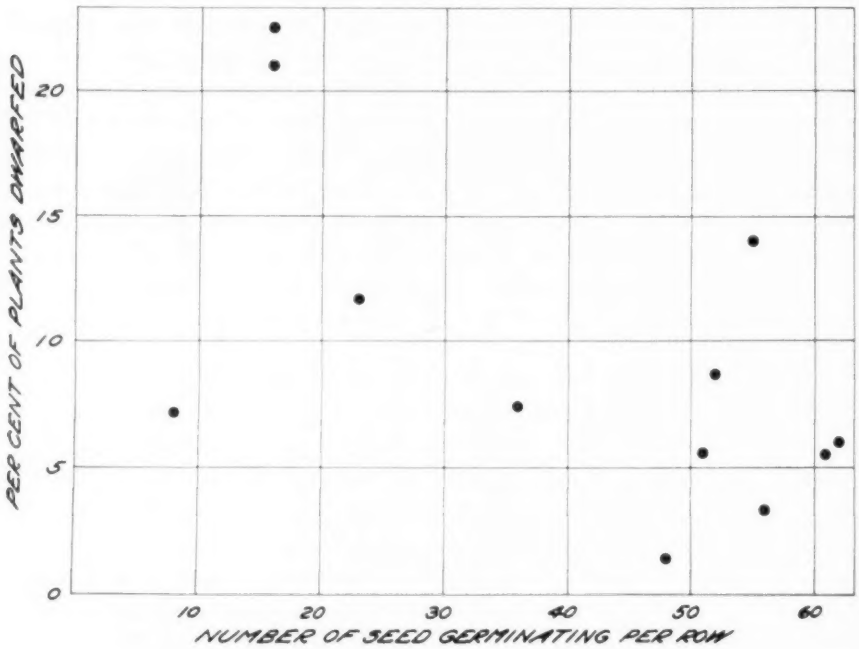

FIG. 1. The relation of germinative vigor to the incidence of pale dwarf in peanut seedlings of the Holle type, grown from seed received from East Java. Each point represents a seed lot from a different locality, grown in systematically replicated 2-rod rows.

IMPORTANCE

Pale dwarf plants were found everywhere in the West Java peanut districts, on both irrigated and unirrigated land and in both the East and the West Monsoons. In the plantings by the natives, the dwarfing was nowhere observed in sufficient amount to constitute a source of real loss. Of numerous plantings made in the writer's experimental work, in only four was it present to a sufficient extent to affect yield. Even in the experimental planting in which it was most prevalent, only one of the 32 seed lots with normal germination vigor produced as many as 10 per cent of plants with undoubted dwarf symptoms. The percentages of plants affected in each of the seed lots in this test are shown in figures 1 and 2. The dwarfed plants are scattered through the plantings. The loss or suppression of such scattered plants in the juvenile stage is approximately equivalent to an early thinning of the stand, as plants so much affected as not to produce a reasonable crop are also too small to have a material competitive influence on the rest of the stand. The space and materials which the dwarfed plants should have utilized are taken by the spreading out of neighboring plants, the spacing in Java peanut fields being much closer than is customary with

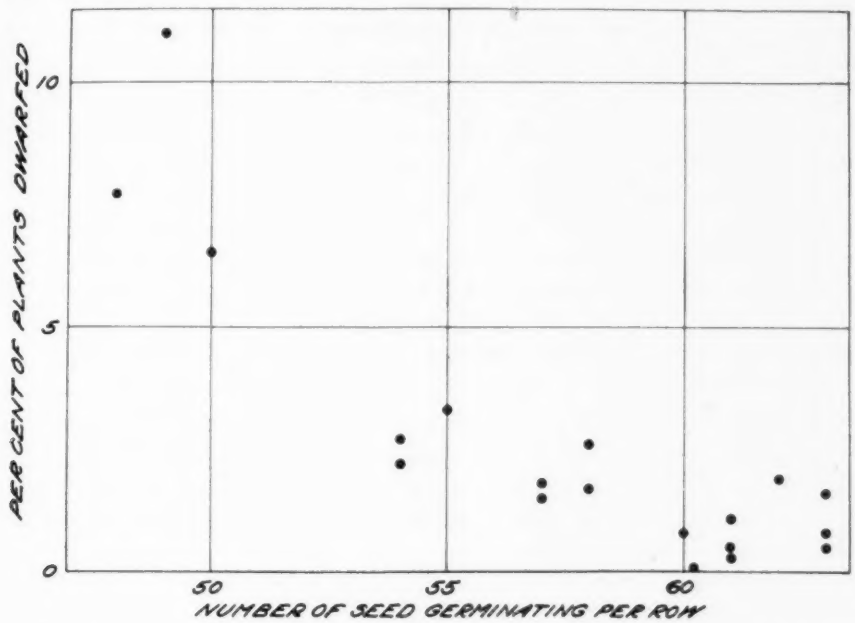

FIG. 2. The relation of germinative vigor to the incidence of pale dwarf in peanut seedlings of the Holle type, grown from seed received from parts of the Netherlands East Indies other than East Java. Each point represents a seed lot from a different locality, grown in systematically replicated 2-rod rows.

varieties of similar growth habit in the United States. Only in cases in which the stand is already unusually thin as a result of seed rot or drouth would the serious dwarfing of 10 per cent of the plants result in a reduction in the ultimate yield approaching 10 per cent.

There is another possibility to be considered in judging the importance of the disease; the association of poor germination with a high percentage of visible dwarfs may mean that the most seriously affected seedlings are kept from appearing above ground, or that the factor which causes pale dwarf can also prevent germination.

DIFFERENCES BETWEEN DIFFERENT SEED LOTS IN LIABILITY TO DWARFING

Tests were made with numerous seed lots of the two standard types of peanut, Holle and Broel, erect-growing types, which mature in 100 days in Java; the Broel has small pods and otherwise closely resembles the so-called Spanish variety grown in the southern United States. The incidence of pale dwarf in the two types was about equal. The same dwarf condition occurred in plants from seed coming from Menado, southern and central Sumatra, and all parts of Java. In addition, there were tested two lots each of the late-maturing prostrate type Tjina, and of a West African peanut intermediate in habit between the erect and prostrate Java types. Both of these types also produced distinct cases of pale dwarf, pictured for the African type in Plate X, B. So far as could be told from the small representation, the two latter types were quite as inclined to dwarfing as the Holle and Broel.

Within the Broel type, in the writer's experiments, the difference in dwarfing tendency between different seed lots was not great. This is in accord with the high degree of constancy found for the different Broel seed lots in the other characters for which they were compared. Within the generally more variable Holle type, it was very evident that different seed lots differed also in dwarfing tendency.

The differences between Holle seed lots in the percentages of dwarfs which they produced were apparently related to the germinative vigor of the seed, and to hereditary differences between different lots. The relation between dwarfing and germination will be considered in the next section. In the present section, comparisons will be limited to seed lots of approximately equal germinative vigor.

It was evident from the results of the experiment in which dwarfing was most prevalent that seed lots received from the Besoeki and Ngandjoek districts, East Java, were decidedly more inclined to produce dwarfs than were the seed lots from other districts. This can be seen by comparing the height of points of equal abscissal positions, figures 1 and 2. A number of these

East Java seed lots had deep red seed skins, and 3 to 4 seeds per pod. It seems more reasonable to attribute the high dwarf tendency of these seed lots to hereditary predisposition than to differences in the conditions under which the seed was grown, in view of the fundamental similarity in climate between the Besoeki and Ngandjoek districts and the Mid-Java districts, from which a large number of the other lots in the experiment had been obtained.

Another indication of hereditary difference in liability to pale dwarf was found within one of the Besoeki seed lots. It contained both red and brown seed. When sown separately in parallel rows, the brown seed, although germinating better than the red, produced plants, 14.0 per cent of which showed pale dwarf effects, while the red seed produced only 1.4 per cent dwarfed plants. In every one of the 6 replications the brown seed row showed more dwarfing than the median of all seed lots for that replication, while the red seed row showed no dwarfing in 4 replications and, in the 2 remaining replications, less dwarfing than the median of all seed lots. This case is evidence that liability to dwarfing is not necessarily associated with red seed skin.

The relative freedom of the Toeban variety was, perhaps, the best evidence of hereditary differences in liability to dwarfing. Of the 25 rows scattered as checks through the experiment represented in figures 1 and 2, 20 contained no dwarfed plants and the remaining 5 practically none. The relative freedom of this variety was further confirmed in 18 rows of Toeban from another seed lot, scattered through another experiment in which were tested 16 additional seed lots not used in the first-mentioned experiment. In this test the Toeban remained entirely free from dwarfing, while 14 of the other 16 varieties were dwarfed. In a still later test, the results of which are shown in figure 3, three different lots of Toeban all showed less dwarfing than did most other lots of equal germination vigor. The percentages of germination of the three lots were 68, 81, and 91, and the percentages of plants dwarfed for the same lots 1.2, 0.6, and 0.3 respectively.

It seems reasonable to conclude that hereditary differences in liability to the pale-dwarf condition are not great, either as between different types, or between different seed lots within the Broel and Holle types. Within the Holle type there appear to be some differences, but much less marked than the hereditary differences, which are found within this type, in morphology and in resistance to *Bacterium solanacerarum*.

No evidence was obtained of transmission through the seed. Seed was obtained from 10 plants that had shown definite dwarfing in early life, and 15 others that had been marked as slightly or doubtfully dwarfed. This seed was sown in cool moist weather. In the 68 plants resulting, not a single case of dwarfing occurred.

RELATION OF SEED VIGOR TO DWARFING

The relation between germination and the incidence of pale dwarf in the seedlings was clearly seen for the Holle type in the results of two experiments shown graphically in figures 1–3. The Broel lots tested in experiments in which pale dwarf was prevalent were so nearly on a par in the matter of germination that no clear relation is evident. The position of the dot farthest to the left in figure 3 supports the expectation that in Broel, as well as in Holle, old seed of low germinative vigor will show the most tendency to dwarfing. In all three graphs the relation appears to be curvilinear.

RELATION OF PHYSICAL FACTORS AND DWARFING

Since the heredity and lack of vigor of the seed sown seem to be only loosely associated with dwarfing, it is necessary to look elsewhere for the real cause of the disease. As no good reason was found for supposing the trouble to be parasitic, the physical environment during the germination period was considered. Abundant dwarfing was found only in plantings which germinated during dry weather; the series showing the largest amount of dwarfing was, furthermore, one in which the seed had been covered less deeply than is usually done. This last finding is in disagreement with the belief of some of the agriculturists in Java, that the dwarfing

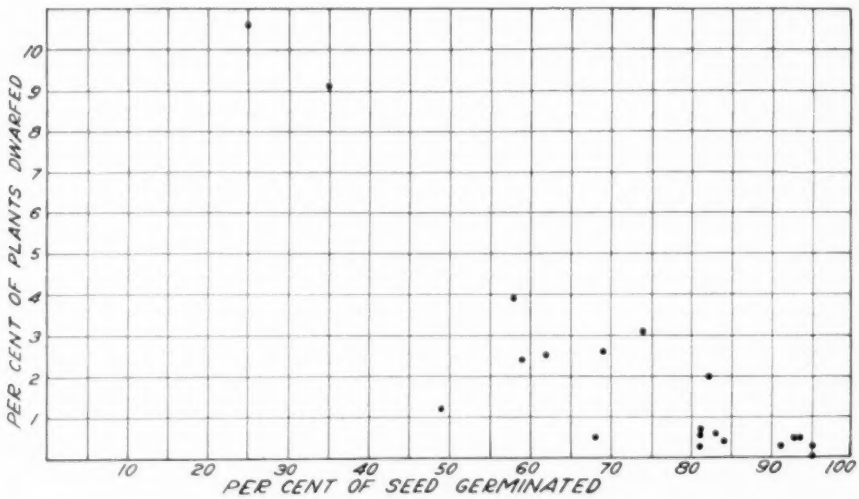

FIG. 3. The relation of germinative vigor to the incidence of pale dwarf in a later experiment. Each point represents a seed lot which was grown in 20 systematically distributed rod rows. The point farthest to the left represents a Broel type seed lot; the others were all of the Holle type.

is due to too deep planting. Both drouth and shallow planting are likely to result in unusually high temperature of the soil and seed during the germinating period. It is suggested that the direct cause of the dwarfing may be prolonged exposure of the sprouting seed to abnormally high temperatures, and the consequent breaking down or inactivation of some substance essential for the growth of the leaflets.

Further indication of heat relation was found in an experiment in which 10 plots were given especially deep cultivation, which resulted in very loose soil at the time of sowing. Ten other plots alternating with them were given little cultivation, and the surface soil was much more compact. As would be expected, the surface soil temperatures were higher in the loose-soil plots because of their poor conducting capacity. Counts showed 118 decided cases of pale dwarf in the loose-soil plots, and only 73 in the compact-soil plots.

Brief heating of the still-dormant seed does not, of necessity, result in dwarfing. In one of the experiments the seed was kept in glass jars during the sowing operations, and exposed to the direct rays of the sun. The seed skins on the side next to the glass soon became discolored, apparently because of heat, and the seed became very hot to the touch, but the amount of pale dwarf in plots sown with this heated seed was actually less than in the plots sown before the seed in the jars had become overheated.

In one experiment, dwarfing appeared in plants which were shaded by trees during a considerable portion of the day. This does not necessarily overthrow the overheating hypothesis. In the United States, pine seedlings have been killed outright by excessive heat, both in nursery beds (3) and forest plots (7), in soil which was exposed to the sun during only part of the day; and in German East Africa, partly shaded plants of *Cedrela odorata* developed typical heat lesions (5).

A somewhat similar case has been described in another plant species. In potato the spindling sprout disease was at one time thought to be due to overheating (6); it may also occur as a symptom of one of the infectious degeneration diseases. In the cereals, Crocker (2) quotes Haberlandt, without citation, as showing that the optimum germination temperature for wheat is 25° C., so far as germination itself is concerned; but credits Gutzeit with showing that, with many small grains, germination at temperatures above 20° C. gives weak plants and small yields, and that the optimum germination temperature from the standpoint of the ultimate yield is 17° C. It seems reasonable to believe that the attention which has been recently given to the killing of plant tissues by excessively high temperatures may well be extended to the question of harmful effects in cases in which there is no obvious necrosis.

Although the type of dwarfing here described has not been reported outside of Java, its presence in seedlings from African-grown seed gives reason for supposing that it will be found in countries other than Java if a search is made for it under proper environmental conditions. The possibility is not excluded that the disease is a manifestation of some systematic parasite which is able to produce recognizable symptoms only in young plants and under special environmental conditions. The probability is, however, that the trouble is entirely non-parasitic.

SUMMARY

1. A new juvenile disease of *Arachis hypogaea* is described under the name "pale dwarf." It is characterized by paling and marked reduction of the leaflets and, in extreme cases, reduction of the petioles. Roots, hypocotyl, and stipules are not dwarfed. Plants usually recover in later life, and mature at the normal time, but as a result of the early setback, their yield is reduced.

2. The disease is generally distributed in West Java, but is usually infrequent and of little economic importance. It has also appeared in seedlings grown in Java from seed brought from West Africa.

3. The incidence of dwarfing is consistently higher in the progeny of some seed lots than in others. This is partly due to the inverse correlation between germinative vigor and dwarfing tendency, but in part probably due to hereditary differences.

4. The dwarfing is believed to be non-parasitic in origin. There is some reason to suspect that it is due to excessive heat of the surface soil after seed sowing; it is suggested that this may destroy or inactivate some substance needed for the normal growth of the leaflets.

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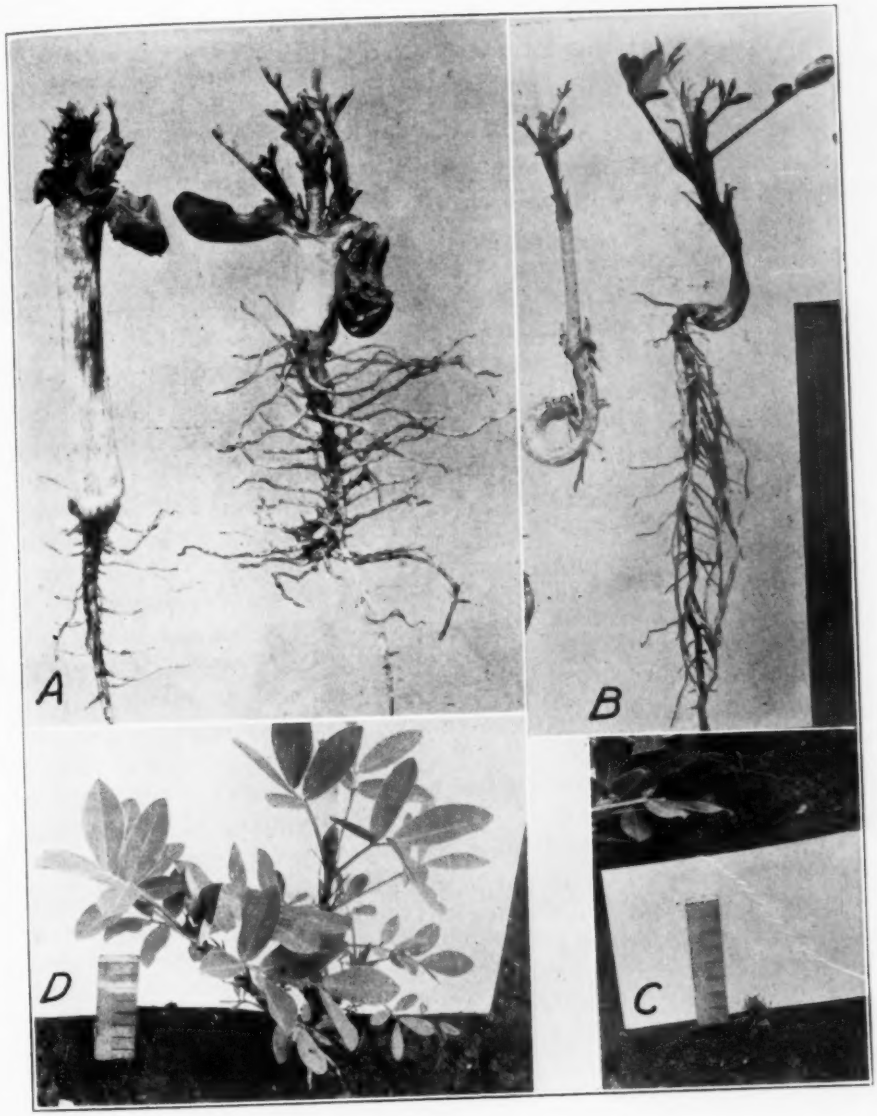
EXPLANATION OF PLATE X

A. Two extreme cases of pale dwarf, from Java Holle seed.

B. Typical pale-dwarf plants, grown from seed imported from west Africa. The near-normal leaves on the right hand plant are on side branches, and were formed later than the much reduced leaves on the central stalk. The black strip shown for comparison is 1 cm. wide.

C. The extremely dwarfed plant in front of the card was less than 2 cm. high, although the photograph was taken six weeks after planting, and numerous pairs of leaves had developed. Although slightly larger leaves were beginning to appear, the plant decayed a few days later, presumably because of lowered resistance due to starvation. Leaves of a normal plant appear at the upper corner of the picture. The stake is marked in centimeters.

D. Holle plant which is producing near-normal leaves after being moderately but distinctly dwarfed in its early stages.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UNIV. OF MICH.

A DISEASE OF COTTON ROOTS PRODUCED BY FUSARIUM MONILIFORME SHELD.[1]

NAOMI CHAPMAN WOODROOF[2]

INTRODUCTION

During the summer of 1920, and again in 1921, the agronomist at Georgia Experiment Station called attention to the very large percentage of dwarfed cotton plants in the fields. Such plants when pulled showed discoloration and in most cases a distinct girdling just below the surface of the soil. Specimens were received from Mississippi during the summer of 1921 with the statement that a very large percentage of the plants were thus affected and were setting few or no bolls.

The trouble was at first attributed to *Rhizoctonia*, but the appearance of the diseased area and the persistence of the dwarfed condition caused some doubt as to the correctness of the diagnosis, since plants attacked by *Rhizoctonia* are either killed outright or else recover and overgrow the lesions during the early stages of growth. It seemed likely, therefore, that some other organism might enter the Rhizoctonia lesions and produce the symptoms noted.

A number of dwarfed plants were brought to the laboratory during July, 1921, and the diseased areas examined microscopically for the presence of *Rhizoctonia*, but this fungus was not found in any case. Seventeen of the plants were placed in a moist chamber for 48 hours. At the end of this time examination showed a growth of Verticillium-like conidia and conidiophores over the surface of the lesions. A culture of this fungus was obtained and in preliminary inoculation tests was found capable of infecting healthy roots of seedling cotton plants.

The following year a more extensive study was planned. A number of fungi were isolated from the surface of the diseased plant roots. Further isolations were made during the summers of 1924 and 1925. Nearly 100 isolations were made from seedlings and older cotton plants.

INOCULATION TESTS

Pot Cultures. All preliminary inoculation tests were made in 6-inch pots of sterile soil which had been autoclaved at 20 pounds pressure for from two to three hours. Four pots were used for each test; in each pot 15 seed were planted. Four pots planted with sterile uninoculated seed

[1] Paper No. 20, Journal Series, Georgia Experiment Station.

[2] The work was carried out under the direction of Dr. B. B. Higgins, who made all notes and observations from 1920 until the summer of 1924.

were included as checks in each series of twelve pots. Thus there was a series of checks for each two groups of inoculated pots.

The seed were prepared for inoculation by delinting with sulphuric acid, removing as much as possible of the acid by washing in tap water, neutralizing the remainder with sodium carbonate and soaking in a 1–1000 solution of bichloride of mercury for 30 minutes followed by two washings in sterile water. The seed were inoculated by shaking in a spore suspension of the desired culture.

Fifty inoculation tests were made using thirty-three different cultures. Twenty-five additional tests were made but discarded in this summary either because they were examined before definite symptoms of the disease had had time to develop or because the check pots became contaminated.

For examination, the soil was washed from the roots of the plants, preserving intact as many of the lateral roots as possible. Counts were recorded and microscopical examinations made. Reisolations were usually made.

It was found that distinct dark brown lesions accompanied by typical girdling do not develop until the third or fourth week following planting.

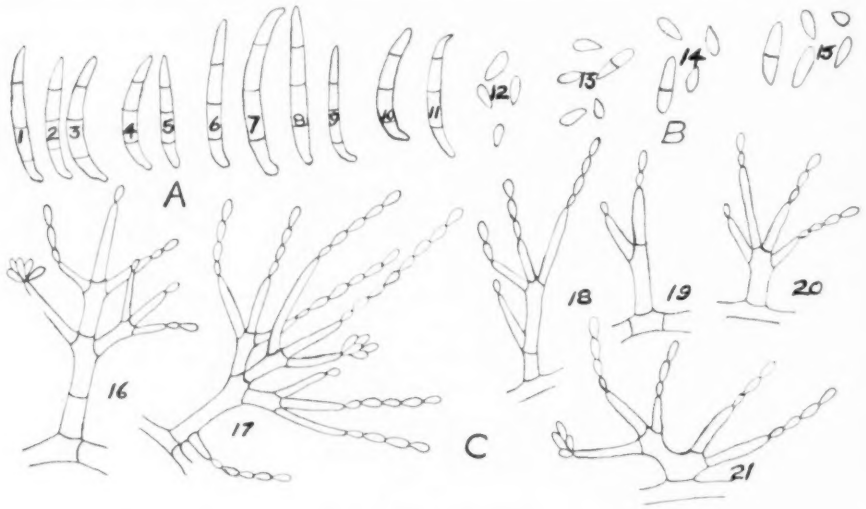

FIG. 1. Conidia and conidiophores of *Fusarium moniliforme* Sheld. Drawn with aid of camera lucida with a Zeiss 4 ocular and a B. and L. 4 mm. objective. A. Macroconidia: 1, 2, 3, from rice after 26 days; 4, 5, from Irish potato plugs after 18 days; 6, from bean pods after 5 days; 7, 8, 9, from Irish potato plugs after 9 days; 10, 11, from bean pods after 16 days. B. Microconidia: 12, from Irish potato plugs after 9 days; 13, from oat agar after 7 days; 14, 15, from bean pods after 12 and 9 days respectively. C. Conidiophores: 16, from cotton stems after 27 days; 17, 20, 21, from water agar; 18, 19, from potato agar after 20 and 7 days respectively.

Yellow discoloration and light brown initial lesions appear by the end of the second week. Figure 2 illustrates the difference in size and appearance of the healthy and diseased plants from the pot tests.

Eight of the thirty-three cultures tested were actively parasitic, all being capable of producing from 90 to 100 per cent infection. The remaining twenty-five cultures were either non-parasitic or only moderately so. All pathogenic cultures produced Verticillium-like microconidia and conidiophores.

Bottle Cultures. Air-borne spores became a serious source of contamination after diseased plants had been grown in the greenhouse in pots for a period of time, even though the benches were thoroughly washed each time with a 1–1000 solution of $HgCl_2$. It became impossible to produce clean checks. In order to overcome this difficulty, bottles of about 4-liters capacity were used. These were filled with moist soil to a depth of about 6 inches, plugged with cotton and autoclaved at 20 pounds pressure for one hour on two successive days. Seed were inoculated in the same manner as for the pots. Fifteen seed were planted per bottle. Four bottles were used for each culture and four uninoculated checks for each group of twelve bottles. Watering was not necessary, as the soil dried very slowly.

The disease developed somewhat more rapidly in the bottles than in the pots, but the symptoms in both were similar. The progress of the disease was observed on roots of plants growing along the sides of the bottles. The yellowing of the primary root and the destruction of the root hairs was followed by a period of a few days to two weeks during which time no visible growth was made by any part of the seedling. Finally, clean, white, lateral roots were pushed out from the discolored surface of the primary root and growth was resumed, but the plants remained smaller than those in the check bottles. In like manner the lateral roots became infected after the development of the root hairs. Thus the usual rapid extension of the root system was prevented.

Field Inoculations. In the spring of both 1925 and 1926 inoculated seed were planted in the field in order to study the effect of heavy infection under field conditions. The tests of 1925 were unsuccessful, owing to the severe drought which prevailed throughout the summer. Only a few seed germinated.

In 1926, spore suspensions of three cultures were used for inoculating two rows of delinted and sterilized seed and two rows of undelinted and unsterilized seed for each of the cultures. Seed similarly prepared but uninoculated were planted as checks. The inoculated seed were dried before planting.

Heavy inoculation failed to increase greatly the percentage of infection over that of the seed treatment tests in which the seed were not inoculated.

There was little difference between delinted and undelinted seed; although the checks showed a slightly higher percentage of healthy plants.

One month after planting, 200 plants from each two rows were dug, washed, and examined. The counts are given in table 1.

TABLE 1.—*Results of inoculating delinted, sterilized cotton seed and undelinted, unsterilized seed with three different cultures of Fusarium isolated from diseased cotton*

Culture no.	Seed delinted and sterilized		Seed undelinted and unsterilized	
	Pct. plants healthy	Pct. plants diseased	Pct. plants healthy	Pct. plants diseased
40	42.5	57.5	50.5	49.5
61	34.5	55.5	28.5	71.5
64	29.0	71.0	25.0	75.0
Check	.....	.....	64.0	36.0

Nos. 64 and 61 were single spore isolations of a culture from an initial epidermal lesion, No. 61 from a typical macroconidium and No. 64 from a typical microconidium. No. 40 was also a single spore isolation from a lesion on the epidermis of seedling roots. This culture was found in the pots and bottles to be very pathogenic and was used as a standard of comparison for other cultures.

SOURCES OF INFECTION

Early in these investigations it was concluded that spores borne on the lint of the seed were a common source of infection to young cotton seedlings. Plants from undelinted, unsterilized seed in sterile soil had a high percentage of infection, while there were few or no lesions on the roots of young seedlings from both delinted seed and delinted, surface-sterilized seed. A pink boll rot, which is common during damp rainy weather, was also found to be caused by the fungus attacking the roots. Therefore, conidia are in all probability present on a high percentage of the lint which appears clean.

The mycelium grows in the soil and is undoubtedly a source of infection for otherwise healthy seedlings. The importance of soil infection will be discussed in connection with the work on control.

As a boll rot is caused by the same fungus as that attacking the roots, the possibility of the mycelium being borne within the seed was considered and a number of tests made. Plants grown in sterile soil from both delinted and delinted, surface-sterilized seed indicated that internal seed infection is relatively unimportant. Plating surface-sterilized seed on agar also

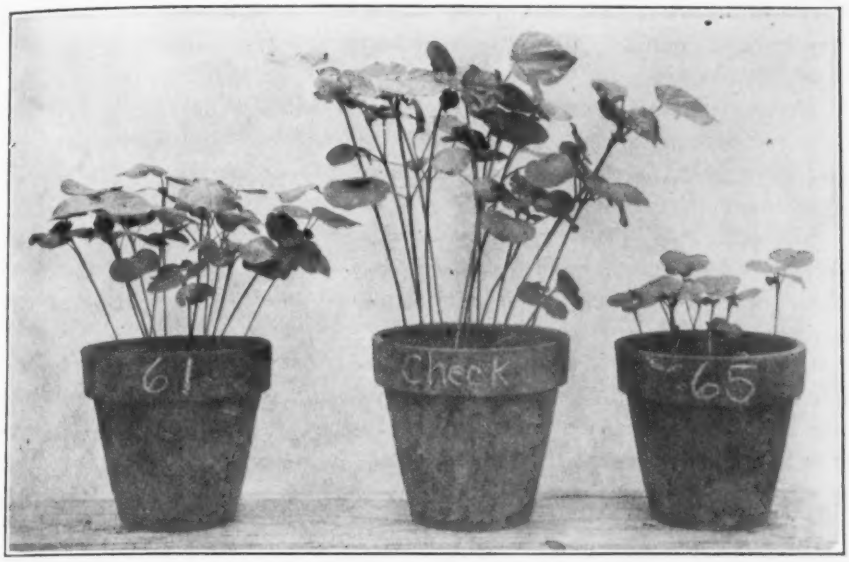

FIG. 2. Cotton seedlings, 24 days old, from disinfected seed (check) and from seed inoculated with two strains of *Fusarium moniliforme*.

showed that in the average lot of seed this is practically a negligible factor. The fungus may be present in the seed of badly rotted bolls but, owing to the difficulty of surface sterilizing such seed and the great number of organisms associated with rotted bolls, this phase of the problem has not been fully worked out.

DESCRIPTION AND IDENTIFICATION OF THE CAUSAL ORGANISM

The following media were employed in the study and identification of the causal organism: steamed bean pods, steamed Irish potato plugs, Irish potato agar (2 and 5 per cent dextrose), oat agar, and steamed cotton stems. The first four media were prepared according to the recommendations of Sherbakoff (4) and the Committee on the Taxonomy of the Fusaria (6).

Ovoid-fusoid and ellipsoid microconidia are produced in chains on verticillately branched conidiophores, ternate branching being the rule.

A few macroconidia are usually present in all cultures and in scrapings from diseased roots, but only in very limited numbers. However, the number of macroconidia is a variable character. Transfers from cultures producing few macroconidia, as well as a few original tissue isolations, occasionally yielded them in abundance. Single spore isolations tend to produce few or many macroconidia according to the type of conidium isolated.

Macroconidia when produced in abundance are borne in sporodochia, which vary in color from a light ochraceous salmon to a light vinaceous cinnamon (2). Macroconidia are with few exceptions three-septate.

Neither chlamydospores nor sclerotia were noted in any of the cultures. Dark bodies on potato plugs, which appeared to be sclerotia under the hand lens, were found by microscopic examination to be merely dark masses of mycelium, not true sclerotia.

The color and quantity of mycelium varies to a considerable extent. The most common color is a pale flesh or shell pink (2). A pink growth of mycelium and conidiophores may often be seen on the surface of diseased roots.

On the starchy media, shades as dark as a slate violet are characteristic of a number of cultures which are otherwise of the usual type, capable of infecting cotton seedlings. The substratum on which the violet cultures grow becomes dark in contrast with the lack of coloration in the substratum on which the pink cultures are grown.

A fluffy, moderately profuse growth of mycelium is most common in culture, but in a few instances a very suppressed mycelial growth is characteristic.

The moniliform method of bearing microconidia on verticillately branched conidiophores, the absence of chlamydospores, and the three-septate macroconidia indicated that the fungus was a *Fusarium* of the section Moniliforme (Liseola of committee) (6). See Fig. 1. According to the descriptions and keys given by the Committee on the Taxonomy of Fusaria (6) and those given by Stevens (5) and comparing the organism with Sheldon's description of *Fusarium moniliforme* (3) it seemed to be identical with the species from corn. Cultures of *Fusarium moniliforme* Sheld., secured through the kindness of Dr. C. D. Sherbakoff, confirmed this conclusion.

METHOD OF ATTACK

Diseased and healthy roots of young cotton plants were prepared and sectioned for microscopical study in order to determine, if possible, the method of attack and the depth of penetration of the mycelium of the fungus. Free-hand sections of the older roots stained with eosin and mounted in glycerine were found satisfactory for studying the more advanced stages.

The mycelium apparently never enters the living cells of the root, as sections have not been obtained showing the mycelium within the cells. A layer, one or two and sometimes three dead cells in depth, may be seen about the periphery of the root. All or only portions of a root surface may

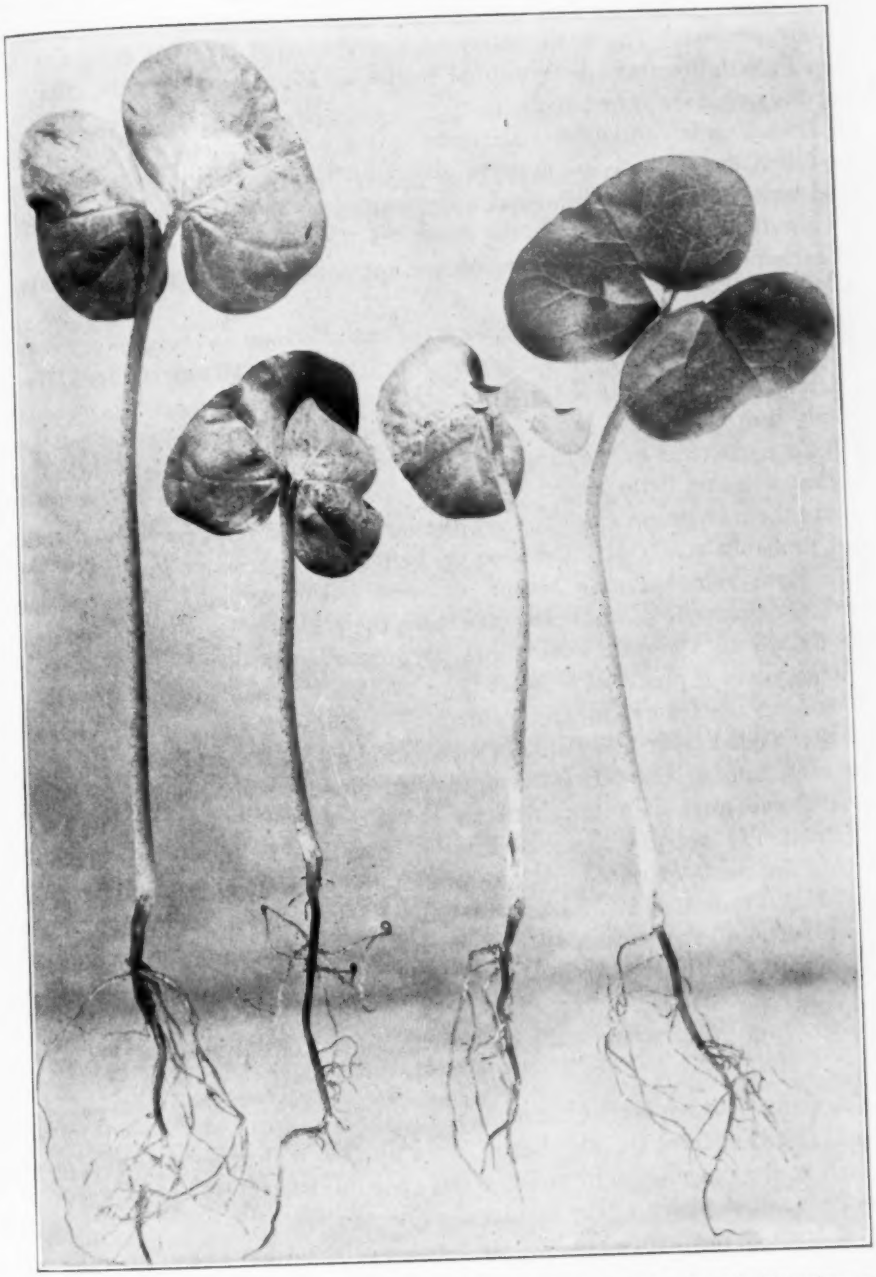

FIG. 3. Cotton seedlings from bottles planted with seed inoculated with Fusarium culture No. 40, showing girdled tap roots. Photographed 16 days after planting.

be affected. After the first three or four weeks the roots are usually completely girdled. The dead, collapsed and darkened layer of cells may be seen in both cross and longitudinal sections. Figure 3 shows the effect of the disease on the young roots.

Dwarfing is caused by a dry, and not a soft, root rot. A soft rotted condition has never been observed either in the inoculation tests or in the field unless associated with some other organism. Pratt (1) reports a soft rot produced by *F. moniliforme* Sheld. on the radicals of seedlings grown in germination tests. Such a condition has not been observed in the course of this work.

The mycelium persists throughout the entire season on the primary roots. *Fusarium moniliforme* Sheld. has been isolated from bits of the bark from primary roots as late as October 15. However, the fungus never penetrates deeply into the cortical layer. The spread of the disease to the lateral root and the persistence of the fungus on the primary root dwarf the entire plant so that it makes little growth.

As the root grows and expands, the layer of dead cells cracks and splits and probably nearly all of it becomes loosened. Successive layers beneath each layer removed may become diseased. However, the whole process probably proceeds slowly. In some cases the plants even seem to overcome the disease to a certain extent and produce various degrees of dwarfing. During years of plentiful rainfall, few or no plants are girdled to the extent of causing the formation of a knot at the surface of the ground. In dry years the dead layer of cells becomes hard, is not easily ruptured but binds the root, causing the definite symptoms of girdling evidenced by the constricted root with a swelling directly above.

Pratt (1) reports that she invariably found *F. moniliforme* Sheld. within the tissue of rotted embryos of cotton seed from Queensland. Such seed failed to germinate. Miss Pratt also reports successful inoculation of green bolls and ripe cotton seed in which she was able to duplicate the condition found in the seed from Queensland.

INOCULATION TESTS WITH CULTURES OF FUSARIUM MONILIFORME SHELD. FROM OTHER SOURCES

A series of three bottles for each of the three cultures of *F. moniliforme* Sheld., secured from Dr. Sherbakoff, were planted with inoculated seed and placed in the greenhouse in order to test their pathogenicity on the roots of seedling cotton plants. The results are given in table 2.

A large percentage of the plants became infected but not so severely as the plants infected by the strains isolated from cotton. The development of lateral roots was not checked to any great extent and the roots were

not so completely girdled. The mycelium of all cultures grew profusely in the soil.

TABLE 2.—*Results of inoculating cotton seeds with three cultures of Fusarium moniliforme Sheld. from different sources*

Culture no.[a]	Per cent plants infected	Per cent plants healthy
R 53	62.8	37.8
R 57	90.3	10.7
1012,3i	93.5	6.5
Check	0.0	100.0

[a] No. R53 was isolated from soil in Central America.

No. R 57 is *F. moniliforme* v. *maius* from soil in Central America.

These two cultures were isolated by Dr. O. Reinking and determined by Dr. Wollenweber.

No. 1012,3i was isolated by Dr. Sherbakoff from germinating corn seed.

CONTROL

In conjunction with the study of the dwarfing disease of cotton, possible measures of control were also considered and numerous disinfectants tested. In view of the disagreeable nature of the standard method of delinting with sulphuric acid and soaking in bichloride of mercury, attention was given to the simpler method of dusting the seed. Forty-five different dusts, including commercial dusts and several combinations prepared in the laboratory, were applied at different rates to the undelinted seed.

Germination tests of all dusted seed were first made. Four hundred seed were used for each test, the effectiveness of each dust was measured by the vigor and readiness with which the seed germinated, the percentage of germination and the comparative freedom from mold and rot of the radicles. Comparisons were made with undelinted checks and with the standard bichloride of mercury treatment. The germinator was kept at 25° to 28° C.

Treatments giving the highest percentage of clean seedlings in the germinator tests were used in dusting seed to be planted in beds in the greenhouse. Germination and freedom from root rots were considered in judging the value of the treatments.

Fifteen treatments including an undelinted and a delinted check and the standard mercuric chloride treatment were selected, on the basis of the greenhouse tests, for trial under field conditions. Field tests have been made in two successive years. The 1925 results were discarded on account of the severe drought. The 1926 trials were more conclusive, but it will be necessary to repeat these under other weather conditions. The planting

season of 1926 was followed by a week of cold wet weather, which delayed germination. The results are summarized in table 3.

TABLE 3.—*The percentages of germination of cotton seed in the field and germinator tests, the percentage of dwarfed plants in the field, and the relative effectiveness of fifteen treatments for the control of Fusarium moniliforme Sheld.*

Disinfectant	Rate per bushel	Per cent dwarfed plants in field	Per cent germination in field	Per cent germination in germinator	Freedom from mold and rot
Semesan No. 13U	4 oz.	44.5	32.10	75.10[a]	Clean
Du Pont No. 18	4 oz.	59.0	27.85	85.88	Clean
Du Pont No. 12	4 oz.	60.5	23.45	85.82	Clean
Mercuric chloride dust No. 1	4 oz.	63.0	26.95	84.46	Fairly clean
Mercuric resinate	Saturated[b]	59.0	26.25	87.30	Clean
Uspulun dust No. 3	Saturated[b]	42.0	17.25	85.70	Clean
Corona 640	4 oz.	58.0	24.10	84.10	Fairly clean
Bayer dust	Saturated[b]	46.5	13.80	82.06	Fairly clean
Mercuric chloride and barium carbonate	4 oz.	55.5	24.40	77.85	Fairly clean
Mercurous chloride	4 oz.	56.0	15.30	83.10	Fairly clean
Mercuric chloride dust No. 2	4 oz.	65.0	22.05	84.20	Fairly clean
Copper carbonate	4 oz.	58.0	16.85	84.37	Fairly clean, injured
Delinted	.....	43.0	15.55	.....	.....
Delinted and soaked 30 min. in 1–1000 mercuric chloride	.....	38.5	21.25	82.53	Fairly clean
Check—no treatment	.....	51.5	18.90	80.84	Poor

[a] Results of only one test, while others are averages of several.

[b] All the dust the seed would take up.

The percentage of germination in the field is much lower than that in the germinator. In the germinator, with the exception of Semesan No. 13U and mercuric chloride + barium carbonate, the percentages of germination of treated seed average from 2 to more than 5 per cent higher than in the untreated check. Discarded treatments often gave a high percentage of germination, but in these, as well as the checks, the great number of rotted radicals which were included in the counts disqualified them. The germination percentages for five treatments in the field averaged lower than the check. It is possible that the increase in the others over that of the check may have been due to the beneficial effects of the treatment.

It is possible to disinfect cotton seed with any one of a number of dusts, controlling all lint-borne organisms so that clean vigorous seedlings are secured in the germinator. However, when seed treated in the same manner are planted under field conditions, soil infection becomes a problem. Higher percentages of dwarfed plants were present in the treated than in the check rows. The field in which the 1926 tests were made had been planted to cotton the year previous. The soil was, as a result, probably heavily infested with *F. moniliforme*. Therefore, it is apparently a difficult problem, if not entirely impossible, to control Fusarium root rot of cotton by seed treatment.

Some of the dust treatments tested appear to be fully as effective in disinfecting cotton seed as the delinting and soaking in mercuric chloride. Dusts are cheaper and more easily applied. However, further field tests will be necessary before definite conclusions can be drawn as to the comparative and actual value of treatments for cotton seed. The tests will be continued and detailed results reported in a future publication.

SUMMARY

Dwarfed cotton plants affected by a distinct type of dry root rot have been noted in the fields of the Georgia Experiment Station for a period of years.

Fusarium moniliforme Sheld. has been determined as the cause of the dwarfed condition.

Dwarfed plants usually remain small throughout the entire season, the causal organism being present on the roots at all times.

The disease is spread on the lint of cotton seed and by air-borne spores, both being means of infecting clean soil with the fungus.

A pink boll rot is caused by *Fusarium moniliforme* Sheld.

Only the outer epidermal cells of the root are killed by the fungus, the cortex and central cylinder remaining healthy.

Cultures of *Fusarium moniliforme* Sheld. isolated from other sources infected the roots of cotton seedlings.

Due to soil infestation with the fungus the disease was not controlled by the use of seed disinfectants.

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A SCLEROTIUM DISEASE OF LARKSPUR

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INTRODUCTION

A study has been made of a disease of the cultivated larkspurs (*Delphinium* spp.), which has been observed for some years in gardens and nurseries in and near Madison, Wisconsin. During 1925 about twenty per cent of the larkspurs in one commercial nursery near Madison was destroyed by this disease. Different horticultural types of larkspur, however, showed different degrees of susceptibility. *D. hybridum* Steph., which has a tall stout stem, appeared rather resistant, while *D. "belladonna"* (Hort.), which has a short delicate stem, was apparently very susceptible. Specimens of *Delphinium* and also of *Physostegia*, which had been destroyed by the same disease, were received from Fort Atkinson, Wisconsin. A disease apparently identical with this has been previously reported on larkspurs from a number of other localities [New York (4), Pennsylvania (1, 4), Indiana (4), and New Jersey (4)].

At Madison, in 1925, the symptoms of the disease became apparent toward the end of June. The first indications were the discoloration of the lower leaves and the wilting of the young shoots, commonly followed within a few days by the death and drying up of the entire plant. At this time a white or greyish white mycelium was found girdling the roots and crown, and forming sclerotia on them. The fungus was commonly confined to these parts. However, during wet weather when conditions were favorable for fungus development, the mycelium was found upon the stems for a distance of five or six inches above the surface of the ground (Plate XI, A, B, and C). Sections of stems in which the disease had reached various stages of development showed that the fungus attack was superficial. Sometimes the mycelium was intercellular in the cortex, where it commonly disintegrated these parts and pushed out the superficial layers. When the soil was examined, the mycelium was found in association with even the smaller roots and seemed to be propagating saprophytically along the cracks of the soil. The disease, although found as early as June, did not develop severely or become conspicuous until August. The severity varied with the condition of the soil, particularly with moisture, the disease increasing rapidly after rainfall.

[1] This research was done under the direction of Prof. L. R. Jones, whom the writer sincerely thanks for his helpful criticisms and suggestions.

THE ASSOCIATED FUNGUS IN CULTURE

The associated fungus was isolated and studied in pure culture. The characters resembled somewhat those of *Sclerotium rolfsii* Sacc. as described from tomato by Taubenhaus (3) and as described by Higgins (2). They were also similar to those of *S. delphinii* Welch from larkspur (4). The study of the associated fungus led the writer to the conclusion that the fungus under consideration was not *S. rolfsii*, but one similar to or identical with *S. delphinii*. It will, therefore, be referred to as *S. delphinii* in this paper.

An examination of a plate culture grown at 28° C. on potato dextrose agar for 5 days (Plate XI, D) showed abundant white mycelium developing radially, mainly in strands. These strands were the result of the branching of the mycelium. The branches usually began their growth at an acute angle with the direction of the main hypha. Subsequently the hyphae grew parallel and tended to hold together in rhizomorph-like strands. The hyphae varied in width from 2 to 10 microns, generally being 4 to 8 microns in diameter. Special feeding branches, which were very slender and generally curved, were produced on the culture medium. Clamp connections occurred in the main hyphae, and anastomosis often took place. The cells of the mycelium usually were multinucleate.

After five days the cultures began to form sclerotia, usually at the edge of the plate. The sclerotia were rather spheroidal, somewhat flattened at the base, and 1 to 5 mm. in diameter. Their color was first white, then yellow or buff, and finally dark red or chocolate. As the sclerotia matured the mycelia collapsed and became inconspicuous (Plate XI, E).

Effect of various kinds of media

A study of the characters of the larkspur organism on different media was made. The comparative development of the fungus on 19 kinds of media is given in table 1. The growth of the mycelium and production of sclerotia varied with the medium. On no medium were spores produced.

On some of the media the development of *S. delphinii* was compared with that of two strains of *S. rolfsii* (Wisconsin stock cultures No. 393 and No. 394). A comparison of the mycelial developments showed no significant differences except that the colony of *S. rolfsii* produced a more dense, cotton-like mass of mycelium than *S. delphinii*. However, distinction appeared in size, shape, and number of sclerotia. The sclerotia of *S. delphinii* were conspicuously larger than those of the two strains of *S. rolfsii*. On potato dextrose agar the sclerotia of *S. delphinii* varied from 1 to 5 mm., commonly 2.5 to 3.5 mm. in diameter, while the sclerotia of *S. rolfsii* (No. 394) varied from 0.9 to 2.5 mm., commonly 1.4 to 1.7 mm. in diameter, and those of the

other strain (No. 393) varied from 0.3 to 1.5, commonly 0.7 to 0.9 mm. in diameter.

TABLE 1.—*Comparative development of cultures of Sclerotium delphinii on different media, grown at 28° C. except as noted*

Medium	Mycelial growth after 4 days[a]	Sclerotial formation after 10 days[a]	Diameter of sclerotia in mm.
Potato dextrose agar	+++	+++	1–5
Synthetic agar	+	+	1–2
Soil extract agar	++	0	–
Bouillon agar	+	0	–
Bouillon dextrose agar	+++	+	1–2
Prune agar	+++	+++	1–3
Oatmeal agar	+++	+++	1–3
Cornmeal agar	+++	+++	1–3
Larkspur decoction agar	++	+	1–3
Iris decoction agar	+++	+++	1–3
Horse dung	+++	+++	2–6
Boiled rice	+++	+++	2–15
Boiled potato	+++	+++	2–15
Raw potato	+++	+++	2–6
Larkspur stem	+++	+++	1–3
Water melon[b]	+++	+++	2–4
Tomato fruit[b]	+++	+++	1–3
Pepper fruit[b]	+++	+++	1–3
Citrus fruit[b]	++	+++	1–3

[a] +++, abundant; ++, moderate; +, scant growth; 0, no formation of sclerotia.
[b] Cultures grown at approximately 20° C.

There was also a striking difference in the number of sclerotia produced by *S. delphinii* and by *S. rolfsii;* the larkspur fungus produced fewer sclerotia (table 2). It was found, however, that the size and number of sclerotia of both species could be made to vary with the amount of culture medium used. In determining this relation, petri dishes 9 cm. in diameter were used, in some of which 10 cc. and in others 20 cc. of potato dextrose agar was poured. A greater number of sclerotia of somewhat larger size was produced on the deeper medium. There was, however, always a marked difference between those of *S. delphinii* and *S. rolfsii.*

The sclerotia of *S. delphinii* were variable in shape, usually spherical, slightly flattened or even concave on the under side, particularly when they were large. The surfaces of the sclerotia were very often pitted. In color the sclerotia were dark red or chocolate, conspicuously redder than those of *S. rolfsii* of which both strains were dark brown.

TABLE 2.—*Relative number of sclerotia produced in plate cultures of Sclerotium delphinii, and Sclerotium rolfsii grown at 27° C. for 10 days*

Medium	Amount (cc.)	Number of sclerotia		
		S. delphinii	*S. rolfsii* (No. 393)	*S. rolfsii* (No. 394)
Potato dextrose agar	10	33	226	238
do	20	69	554	276

Effect of temperature

The effect of temperature on the growth of *S. delphinii* in culture was studied in comparison with that on two strains of *S. rolfsii*. The results are given in table 3. Development after one month was as follows: at 5° C. none of the fungi had grown; at 10° C. *S. delphinii* had developed a very scant mycelium, while neither strain of *S. rolfsii* showed any growth; at 14° C. *S. delphinii* had produced mature sclerotia, *S. rolfsii* No. 394 white abortive sclerotia, and *S. rolfsii* No. 393 an abundant white mycelium but no sclerotia; at 18° C. *S. delphinii* and *S. rolfsii* No. 394 had formed mature sclerotia, but *S. rolfsii* No. 393 an abundant white mycelium without sclerotia; at 21° C. *S. rolfsii* No. 393 had produced small sclerotia here and there, and the other two cultures mature sclerotia. Above this temperature all the fungi produced mature sclerotia. The result of this experiment, therefore, showed that *S. delphinii* can grow at a rather lower temperature than *S. rolfsii*. The optimum temperature for *S. delphinii* appeared to be from 28° to 30° C.

TABLE 3.—*Average diameters in millimeters of two colonies each of Sclerotium delphinii and Sclerotium rolfsii grown at different temperatures for four days on potato dextrose agar*

Fungi	Temperature in degrees C.							
	30.5	28	24	21	18	14	10	5
S. delphinii	71.5	71.5	40.5	35.0	4.5	3.0	0	0
S. rolfsii No. 393	64.0	54.0	30.0	21.0	6.5	2.0	0	0
S. rolfsii No. 394	72.0	70.0	31.0	22.5	2.0	2.0	0	0

PATHOGENICITY OF THE FUNGUS

Both gross and microscopic examinations indicated that the disease was caused by the associated fungus (*Sclerotium delphinii*). In order to deter-

mine the relations, inoculations were made on several species of plants with pure cultures of the organism.

In the first experiment three healthy larkspur plants, which had grown for two years in the nursery, were transplanted into pots and ten days later, on July 7, inoculated as follows: the soil was dug from about the stem for a depth of 5 mm. and mycelium from a potato agar culture of the fungus was placed next to the stem and covered with soil. The plants were kept in the moist inoculation chamber for 24 hours and then removed to the greenhouse. The mycelium spread a little, but soon formed sclerotia and no infection occurred. The following facts may explain the negative results: (a) The period in the inoculation chamber was too short; (b) the greenhouse was dry and the pots were watered but once a day. These conditions may have made the soil too dry for the fungus to develop and produce infection.

In the second experiment, on August 13, inoculations as listed in table 4 were made. The inoculation method was the same as in the first experiment except that the plants were kept in the moist inoculation chamber for two days and were then watered twice each day.

TABLE 4.—*Results, after 14 days, of inoculating various plants with Sclerotium delphinii*

Plants inoculated	Age of plants	Number of plants		Number of plants	
		Inoculated	Infected	Uninoculated	Infected
Delphinium hybridum	2 years	2	2[a]	1	0
D. "belladonna"	6 months	3	2	2	0
Bean	29 days	7	0	3	0
Cucumber	do	7	1	2	0
Tomato	do	7	0	3	0

[a] One plant killed. The other plant lost the old shoots but eventually grew new ones.

The inoculations made in the third experiment, on August 29, are shown in table 5. In this experiment the plants were kept in the moist inoculation chamber for 4 days. The subsequent temperature also was somewhat higher than in the preceding experiments, ranging from 25° to 30° C.

The results of these experiments, as given in tables 4 and 5, showed that the fungus was parasitic on the larkspur, and that it might also attack melon, cucumber, and rice if the conditions were favorable for infection. The symptoms on these inoculated larkspurs were essentially like those of the original disease. On the cucumber the fungus invaded and rotted the

stem at the ground line, the decayed parts collapsing and turning a brownish color.

TABLE 5.—*Results, after 10 days at 25–30° C., of inoculating various plants with Sclerotium delphinii*

Plants inoculated	Age of plants	Number of plants		Number of plants	
		Inoculated	Infected	Uninoculated	Infected
Delphinium "belladonna"	6 months	2	2	0	–
D. ajacis	2 months	10	2	2	0
Bean	45 days	10	0	3	0
Cucumber	do	5	2	1	0
Tomato	do	10	0	2	0
Melon	36 days	3	1	1	0
Squash	do	2	0	1	0
Rice	45 days	3	1	1	0

SUMMARY

1. A disease of larkspur is reported from Madison, Wisconsin, and vicinity. Different varieties of larkspur have different degrees of susceptibility to the disease. Cultural studies show it to be caused by a fungus similar to or identical with *Sclerotium delphinii*.

2. The mycelial characters of *S. delphinii* and *S. rolfsii* are similar. The sclerotial characters of these species, however, differ. The sclerotia produced by *S. delphinii* are fewer in number, conspicuously larger, usually more flattened, and redder in color than those produced by *S. rolfsii*. Also, *S. delphinii* is able to develop at a somewhat lower temperature than *S. rolfsii*.

3. The production of spores has never been observed even though the fungus was grown on 19 different media.

4. The depth of the medium influences the size and number of sclerotia produced by the fungus. They are smaller and fewer in number on the thinner substratum.

5. The development of *S. delphinii* is affected by temperature, making but scant growth at 10° C. and apparent optimum growth at 28° to 30° C.

6. The fungus has been found pathogenic to larkspur and, to a lesser degree, to melon, cucumber, and rice.

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EXPLANATION OF PLATE XI

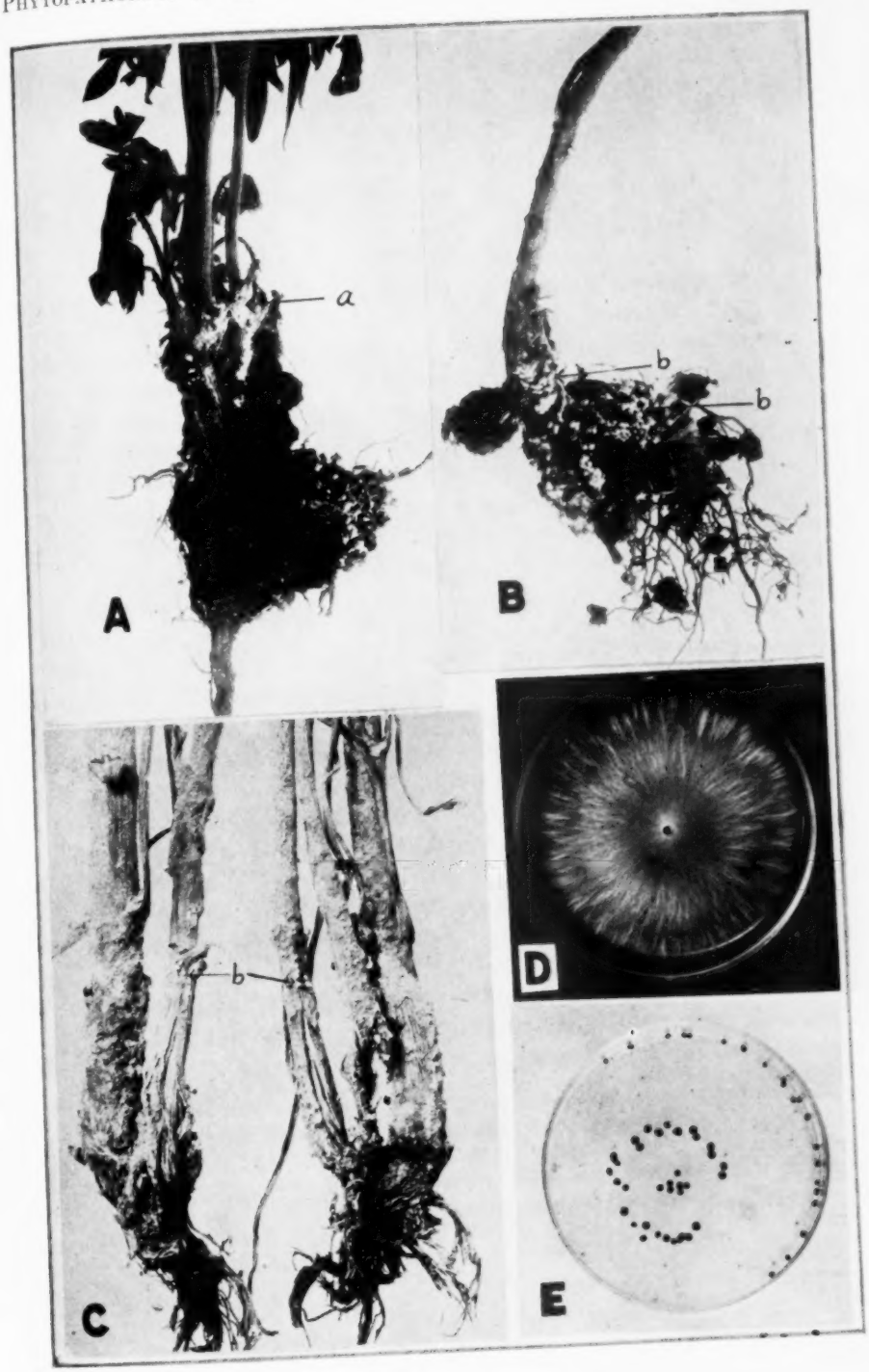
A. Larkspur newly infected by *Sclerotium delphinii*. White mycelium girdling the crown is seen at *a*.

B. Symptoms on larkspur a few days later than A. There are numerous new sclerotia on the roots, a few of which are indicated by the arrows marked *b*.

C. Typical symptoms of larkspur attacked by *S. delphinii*. The roots are almost entirely rotted, and the stem is dying from the base upward. Some sclerotia are seen on the stem at *b*.

D. Development of mycelium on potato dextrose agar after 3 days at 28° C.

E. Formation of sclerotia on potato dextrose agar after 10 days at 28° C.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UNIV. OF MICH.

PHYSIOLOGIC SPECIALIZATION IN TILLETIA LEVIS AND TILLETIA TRITICI[1]

H. A. Rodenhiser and E. C. Stakman[2]

INTRODUCTION

With the increased attention during the past few years to the development of disease-resistant varieties of crop plants, the investigation of the possible rôle of physiologic specialization of the pathogenes involved assumes additional importance. While most of the cereal smut fungi can be prevented fairly well by seed treatment, the method is not always completely effective. In any case, it is desirable to use disease-resistant varieties if they are available. One of the important factors in determining the value of disease-resistant varieties is the degree of parasitic specialization, mobility, and genetic constancy of the pathogene. It is known that there are different physiologic forms of some pathogenes in different regions, and that different forms may be present in the same region in different years (6).

Reed (4), Faris (2), Stakman and Christensen (8), Christensen and Stakman (1), Rodenhiser (5), and Tisdale and Johnston (9) have shown that certain of the smut fungi comprise distinct physiologic forms. It seemed quite likely that the same might be true of *Tilletia levis* Kühn and *Tilletia tritici* (Beij.) Wint., especially since Faris (3) obtained some preliminary evidence of differences in pathogenicity.

In the hard red spring wheat region of the United States, bunt of wheat has not been very destructive during the past ten years. The principal reason seems to be that Marquis and the durum wheats, which are grown most commonly, are resistant. But during the summer of 1925 there was an unusual outbreak of bunt, caused principally, in Minnesota at least, by *T. levis*, although *T. tritici* also was found to some extent. The natural supposition would be either that weather and soil conditions were unusually favorable for the development of bunt, or that there might have been un-

[1] Published with the approval of the Director of the Minnesota Agricultural Experiment Station as paper No. 690 of the Journal Series of the Minnesota Agricultural Experiment Station.

[2] The writers are indebted to the following persons for bunt material: Dr. F. N. Briggs, California; Dr. F. D. Heald, Washington; Dr. B. Husz, Hungary; Dr. E. Pantanelli, Italy; Dr. I. Jorstad, Norway; Dr. T. Lindfors, Sweden; Dr. T. Fahmy, Egypt; Dr. G. H. Cunningham, New Zealand; and Mr. I. L. Conners, Canada.

usually virulent strains of the pathogene. Therefore the writers attempted to ascertain whether there actually are distinct physiologic forms of *T. levis* and *T. tritici*.

MATERIALS AND METHODS

Five collections of *T. levis* and seven of *T. tritici* were obtained in 1925, from the places indicated in tables 1 and 2.

In the spring of 1925 Kota wheat, C. I. 5878,[3] was inoculated with each collection of smut except those from California and Washington, which had not yet been obtained. The smutted heads which resulted were then picked and kept under the same conditions until the spring of 1926. Thus the spores of all of the collections, with the exceptions mentioned, were produced and kept under similar conditions.

Mindum, C. I. 5296, a durum wheat; Einkorn, C. I. 2433; Marquis, C. I. 3641, and Kota, C. I. 5878, both hard red spring wheats, were inoculated with chlamydospores of each collection of smut. Previous to inoculation, the seed was treated by Jensen's modified hot water method. When thoroughly dry, enough seed for duplicate rod rows was inoculated with powdered inoculum from each collection, at the rate of 0.5 grams to 100 grams of seed, and all seed was sown on the same day in duplicate, systematically distributed rod rows. As there was very little difference in the percentage of smut in the duplicate rows, only the averages are recorded.

With one exception, the percentages of infection given in tables 1 and 2 are based on counts of 800 heads of Einkorn, 800 of Marquis, and 1,000 of Kota. The counts on the varieties inoculated with the Egyptian collection are based on counts of 400 heads of Einkorn, 400 of Marquis, and 500 of Kota. The percentages of partially and completely smutted heads of Einkorn and of Marquis were recorded separately. The number of partially smutted heads of Kota was not determined, because there was very little partial smutting. So little smut developed in Mindum that the results are not recorded.

RESULTS

The results of the tests are given in tables 1 to 3 inclusive. The results with Einkorn and Marquis are considered more significant than those with Kota, because the two former usually are so resistant that one would not expect much variation in the percentage of infection. These varieties have been grown at University Farm, St. Paul, for seven years, from artificially smutted seed, and there always has been a high degree of correlation be-

[3] C. I. = accession number of the Office of Cereal Crops and Diseases, Bureau of Plant Industry, United States Department of Agriculture.

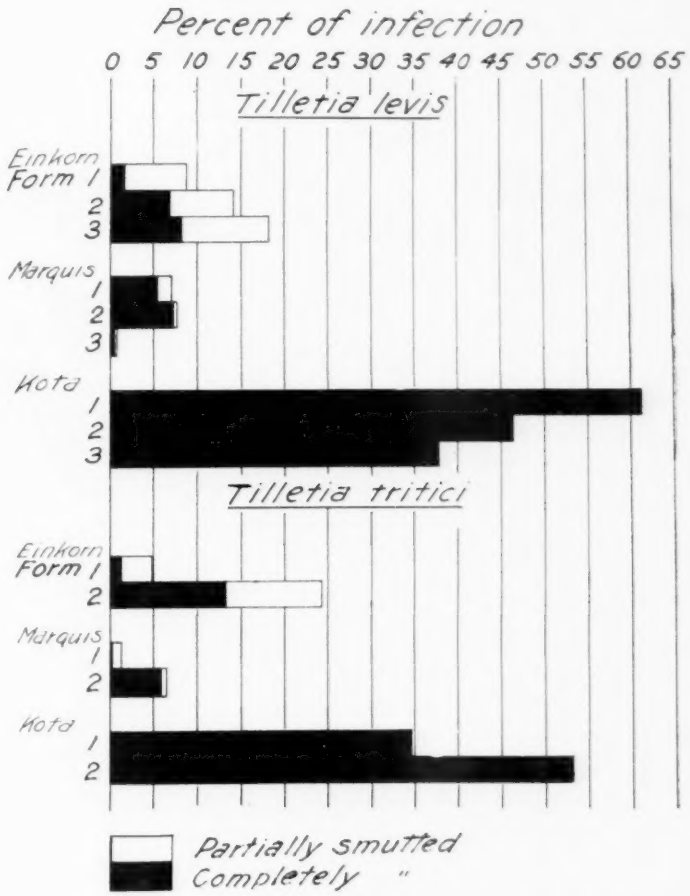

FIG. 1.—The percentages of infection in Einkorn, Marquis, and Kota inoculated with three physiologic forms of *Tilletia levis* and two of *T. tritici*.

tween the percentages of bunt in different years, as well as in replicated rod rows in the same year.

It is evident from figure 1 and tables 1 and 2 that there are sufficiently great differences in the virulence of several collections of smut to justify the conclusion that there are physiologic forms. In table 3 is indicated the relative susceptibility of each variety to each of these forms.

It seems clear from table 3 that there are at least three physiologic forms of *T. levis*. The two collections from Hungary were so nearly alike that they are considered to be identical and are designated as form 1. The collection from Minnesota behaved much like form 1 on Marquis and Kota,

TABLE 1.—*The percentage of smutted heads in Marquis and Kota wheats and Einkorn inoculated artificially with five collections of Tilletia levis at University Farm, St. Paul, Minn., in 1926*

Source of inoculum	Germination of chlamydospores in per cent	Percentage of smutted heads						
		Einkorn			Marquis			Kota
		Partial	Complete	Total	Partial	Complete	Total	Total complete
Minnesota	65	8.4	6.7	15.1	0.4	7.1	7.5	64.3
Hungary[a]	80	12.7	1.9	14.6	0.9	4.6	5.5	61.7
Hungary[b]	75	7.4[c]	1.5	8.9	1.7	5.3	7.0	61.1
Italy	80	13.4	7.3	20.7	1.2[c]	0.2	1.4	56.7
Egypt	90	9.8	8.3	18.1	0.3[c]	0.3	0.6	37.6
Check (uninoculated)		0.0	0.0	0.0	0.0	0.0	0.0	0.0

[a] Hatvan.

[b] Debreizen.

[c] Individual heads lightly smutted. Only a few smutted, or partially smutted, kernels in each head.

TABLE 2.—*The percentage of smutted heads in Marquis and Kota wheats and Einkorn inoculated artificially with seven collections of Tilletia tritici at University Farm, St. Paul, Minn., in 1926*

Source of inoculum	Germination of chlamydospores in per cent	Percentage of smutted heads						
		Einkorn			Marquis			Kota
		Partial	Complete	Total	Partial	Complete	Total	Total complete
New Zealand	80	3.7[b]	1.1	4.8	1.0	0.3	1.3	34.5
Hungary[a]	65	9.5[c]	9.7	19.2	0.4	2.7	3.1	50.4
Norway	70	11.1	13.2	24.3	0.6	5.9	6.5	53.4
Sweden	80	9.9	1.9	11.8	1.8[b]	0.7	2.5	58.0
Manitoba	80	5.8	6.4	12.2	1.0	4.2	5.2	37.5
California	80	10.0[c]	13.7	23.7	1.5	0.5	2.0	40.0
Washington	65	4.0	6.5	10.5	1.4	0.4	1.8	28.5
Check (Uninoculated		0.0	0.0	0.0	0.0	0.0	0.0	0.0

[a] Hatvan.

[b] Individual heads lightly smutted.

[c] Individual heads almost completely smutted.

but was considerably more virulent on Einkorn and therefore is considered as form 2. The collection from Egypt was slightly more virulent on Einkorn than the other two, decidedly less virulent on Marquis, and considerably less virulent on Kota. Therefore it is designated as form 3. The collection from Italy was very similar to the one from Egypt, although apparently less virulent on Marquis and Kota. It may be a fourth form, but, until further tests are made, it is not so considered.

TABLE 3.—*The relative susceptibility, of Einkorn, Marquis and Kota to four collections of Tilletia levis and to three of T. tritici*

Source of inoculum	Relative susceptibility[c]			Form number
	Einkorn	Marquis	Kota	
Tilletia levis				
Hungary[a]	R	R	S	1
Hungary[b]	R	M R	S	1
Minnesota	M R	M R	S	2
Egypt	M R	V R	M S	3
Tilletia tritici				
New Zealand	V R	V R	M S	1
Norway	M R	M R	S	2

[a] Hatvan.

[b] Debreizen.

[c] V R = very resistant; M R = moderately resistant; M S = moderately susceptible; S = susceptible.

One could conclude that there are at least five distinct forms of *T. tritici* (table 2), but no final conclusions are based on the results obtained with the collections from Washington and California, because the inoculum was not produced and stored under the same conditions as those for the other collections. Nevertheless there are at least two forms: form 1, from New Zealand and form 2, from Norway. The New Zealand collection was consistently less virulent than that from Norway and the differences are so great that one is forced to the conclusion that they represent two distinct forms. For the present, the collection from Hungary is considered to be form 2. The collections from Manitoba and Sweden are similar to forms 2 and 1 respectively on Einkorn and Marquis, but both collections differ somewhat in their effect on Kota and it is probable that they can be differentiated more clearly on other varieties of wheat.

SUMMARY

1. *Tilletia levis* and *T. tritici* both comprise distinct physiologic forms which can be recognized by the degree of their virulence on Kota and Marquis wheats and on Einkorn.

2. Collections of *T. levis* were obtained from Minnesota, Italy, Egypt, and two localities in Hungary. There were at least three physiologic forms in these collections: one from Minnesota, one from Hungary, and one from Egypt. The Italian collection possibly may represent still another form.

3. Collections of *T. tritici* were obtained from New Zealand, Hungary, Norway, Sweden, Canada (Manitoba), and from Minnesota, California, and Washington in the United States. Two forms can be recognized readily: a virulent one from Norway, and a relatively weak one from New Zealand. There is evidence that there are still other forms.

4. It seems likely that a considerable number of forms both of *T. levis* and *T. tritici* can be distinguished if the proper differential hosts are used.

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SECOND PROGRESS REPORT ON BUNCHY-TOP OF ABACÁ, OR MANILA HEMP

GERARDO OFFIMARIA OCFEMIA

In the abacá plot of the Department of Agronomy of the College of Agriculture at Los Baños, Laguna Province, Philippine Islands, in specimens collected from Cavite Province, and in transmission experiments of bunchy-top using *Pentalonia nigronervosa* under controlled conditions, it has been noted that in advanced stages of the disease "heart rot" sometimes sets in. This observation is of special significance, because of the attention which heart rot has attracted. According to Mr. Melanio R. Calinisan, who is working on the relation of nematode (*Heterodera radicicola*) to bunchy-top of abacá, the abacá farmers of Cavite Province believe that the heart rot is more important than the bunchy-top. In a conference with Mr. Felicisimo B. Serrano, Assistant Plant Pathologist of the Philippine Bureau of Agriculture in Manila, who has been working on the heart rot of abacá since 1920, it was learned that Mr. H. Atherton Lee, formerly Mycologist of the Philippine Bureau of Science in Manila, considered heart rot as the more destructive of the two abacá diseases. Lee and Serrano[1] reported having isolated a *Fusarium* closely resembling *F. cubense* from cases of heart rot of abacá. These authors claim that they obtained positive results from inoculation experiments using *F. cubense* for the production of abacá heart rot. It is not clear, however, how this vascular *Fusarium* of the banana could produce heart rot on abacá.

In aphid-transmission experiments of bunchy-top of abacá under controlled conditions, it has been noted that the first symptom of the disease is the appearance of yellowish white, indefinite chlorotic areas on the margin of the youngest leaf. The green parts of the leaf blade on each side of the midrib are darker green than the leaves of normal plants. The leaves produced are smaller and show a tendency to curl up along the margin. As the curling of the leaf margin is a more characteristic symptom of the virus form of bunchy-top of abacá than the bunching of the leaves, the disease might better be known as "curly-top." The presence of the greenish yellow or yellowish white areas and its transmissibility by aphids might group the disease with the mosaics or transmissible chloroses. The chlorotic areas

[1] Lee, H. Atherton, and F. B. Serrano. Banana wilt of the Manila hemp plant. Phytopath. 13: 253–256. 1923.

are retarded in their normal growth, and as a result an affected leaf has a tendency to tear along the margin. Very often delicate, thin, transparent, membrane-like areas of different shapes and sizes are present on the chlorotic portions of the youngest leaf. These membrane-like areas are visible before the leaf unfolds or immediately after (Fig. 1, a). When the transparent membrane-like areas are present on the greater portion of the unexpanded

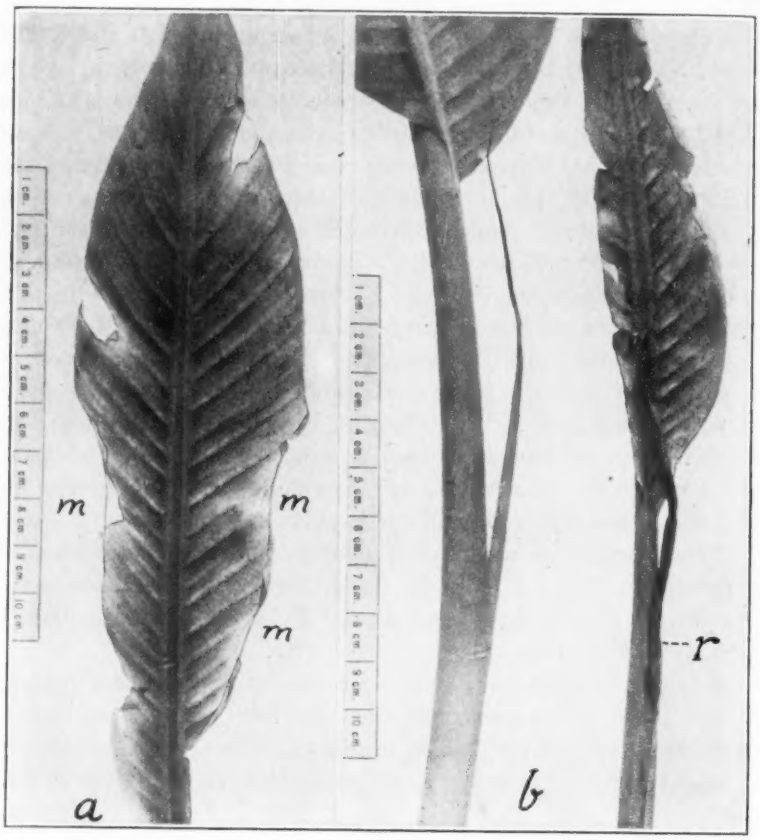

FIG. 1. (a) A young leaf of abacá, C. A. 4293 Itom, affected with bunchy-top from aphid-transmission experiments showing the membrane-like areas (m) along the margin of the leaf. The membrane-like tissues on the upper part of the leaf were torn off.

(b) At the left is shown the youngest leaf of a healthy abacá seedling, C. A. 4293 Itom. At the right is the heart of a bunchy-top-infected abacá seedling from aphid-transmission experiments showing the browning of the youngest leaf (r) and the beginning of the rotting of the brown tissue.

All photographs were taken by the Photographic Laboratory of the Bureau of Science at Manila, P. I., two months after inoculation.

youngest leaf, browning follows, and if weather conditions are favorable rotting sets in (Fig. 1, b), starting from the top and working downwards. In this type of heart rot, bacteria are present in great abundance and hasten the rapid decay of the soft tissues of the heart. This type of heart rot is probably the same as Reinking's bacterial heart rot of abacá.[2]

Although it is not claimed that all heart rots of abacá are secondary diseases, it seems that, in bunchy-top infected districts at least, many of the heart rot cases are probably the final stages of bunchy-top.

In results thus far obtained, it has been noted that the abundance of nematode galls in the roots of abacá, the rotting of roots due to various soil fungi, and close planting may induce bunching of the leaves resembling that of the aphid-transmissible bunchy-top. In bunchy-top brought about by any of these three conditions, however, the chlorotic areas on the leaves, the diminution of the size of the leaf, the curling of the margin, and other malformations are not shown. Further work is in progress.

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[2] Reinking, Otto A. Philippine economic-plant diseases. Philippine Jour. Science Sec. A. 13: 165–274. 1918.